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XLVII

CARE OF MASTOID WOUNDS

FOLLOWING THE COMPLETE MASTOID OPERATION, SINUS THROMBOSIS AND OPERATIONS ON THE PETROUS PYRAMID*

MARVIN F. JONES, M.D.

NEW YORK

From the patient's viewpoint, the convalescent period overshadows the oblivion of the actual operation. Postoperative dressings may be fearsome or they may be done leaving a memory of their accomplishment as a not too unpleasant incident of the day. For the surgeon, a newer concept of adequacy arises. No longer is a good operation sufficient. The surgeon of today is expected to produce a satisfactory functional and an acceptable cosmetic end-result painlessly. The time consumed by convalescence is most important. Absence from work is especially important in these days when a long disability may mean the loss of a job and consequent loss of income. The main features of this paper will, therefore, be painless dressings, cosmetic perfection, restoration of function and a shortened period of disability. The complete mastoid, being the most frequently performed operation in otology, will be the first to be discussed. Since our president requested personal methods, I will describe methods that I have used.

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When the mastoid operation has been completed, Michel clips are used to close the wound, two or three clips being adequate. The largest Sirus drain is placed with one end in the antrum cavity and the other extending through the lower extremity of the wound, protruding for about one-half inch. A plain drain moistened with alcohol and boric acid, equal parts, is packed in the external canal. The outer dressing and drain in the canal are changed daily without disturbing the clips or Sirus drain. On the fourth or fifth postoperative day, the clips are removed and the Sirus drain changed for one of a smaller size. If care is observed when removing the clips, any patient who has not reached the co-operative age or those few patients whom parental influence has rendered unmanageable, must be "mumified." (An inelegant term which is expressive and describes a patient wrapped in a sheet.) The number of times "mumification" will be necessary will be in direct proportion to the patience of the doctor. Rarely have I found it necessary to give an anesthesia for dressings, and this statement includes the much dreaded first complete dressing.

The number of days elapsing between dressings depend upon the amount of discharge. A few need to be dressed daily, but more commonly an interval of one to three days is preferable. The Sirus drain may be loosened and allowed to remain. When the drain is changed a smaller size is reinserted. A gentle twisting movement makes the procedure more comfortable. Drains are used until the external canal is free from discharge. The time of healing is one to four weeks in uncomplicated cases. The postoperative incision closes with a barely perceptible scar and no deformity. I have used the following methods and discarded them: Suturing the wound, tight packing with gauze, rubber tissue or rubber tube drainage and primary complete closure.

Sometimes a wound must be left open. When this is necessary a secondary closure is done some days later and the aftercare following the closure is the same as has just been described. Persistent sinues have been of rare occurrence in recent years, but they offer no problem. A method which I have used some fifteen years has been entirely effective. The mastoid excavation is opened and a wide elevation of periosteum posterior to the wound plus some elevation of the perichondrium anterior produces a skin flap from which the nonviable epithelium is removed. A second incision is made in the scalp paralleling the first. The result is a strip of tissue one or two inches in width attached at each end. The periosteum is then elevated, leaving a freely movable ribbon of tissue. The first

incision is closed with Michel clips and quickly heals. The posterior wound may be closed seven to ten days later, but by this time the posterior incision has usually healed spontaneously.

Sinus and Jugular: This operation evidently will continue to be a controversial procedure as long as surgery is done. The postoperative care has changed little except to obtain better cosmetic results.

My custom has been to do the operation on the sigmoid sinus first, and after changing to an entirely new sterile setup, do the operation on the jugular vein. The reasons for using this method do not enter into the scope of this paper, but it is necessary to mention the method at this point in order to rationalize the aftercare.

The mastoid cavity is left completely open with the exception of a stay suture of silkworm gut. Iodoform gauze tampons are placed over each end of the exposed sinus. The remainder of the cavity is packed with the same material. So much for the mastoid part.

The method of closing the neck wound depends on the patient. If I anticipate strain on the incision, the neck wound is closed with Michel clips. A subcutaneous suture of No. 1 catgut may be used on peaceful patients, and the resultant scar is invisible. A small cigarette drain is placed in the neck structures, parallel to the incision, with its exit at the lower extremity of the wound. Drainage material is removed at the end of 48 hours. If Michel clips have been used, they are removed three days after operation.

The patients who have had subcutaneous catgut used are subjected to no inconvenience after operation. An oiled silk apron is fastened to the neck by collodion. During the neck dressings this is turned up over the mastoid dressing, and when the neck dressing has been finished, it is turned down as a cover and protection against any possible seepage from the mastoid wound.

Provided the progress of the patient is favorable, the sinus dressing is not disturbed until the fourth or fifth day. At this time the gauze packing is thoroughly soaked with peroxide. By carefully removing the gauze and making frequent applications of peroxide, the first part of the dressing can be accomplished without disturbing the wound and with no undue discomfort to the patient. When dealing with children or very nervous patients, the first dressing is done under anesthesia. The next step is to remove the plugs.

Since the jugular vein has been tied and there is less apt to be bleeding from below, I remove the lower plug first. Again the peroxide is applied and the plug grasped firmly in a mouse-tooth forcep. A gentle side to side and rotary traction is usually effective in removing the plug. Provided bleeding does occur, the plug is left in place and removal tried again at daily intervals until the removal is accomplished. The upper plug receives the same treatment. When the plugs have been successfully removed, a secondary closure of the wound is done, and thereafter the treatment is the same as in a complete mastoid. Healing should require about one week to ten days longer than a mastoid.

Petrositis: Various types of operative procedures for petrositis require different methods of postoperative care. The two methods I have used successfully will be the only ones discussed in this paper. Again I refer to the president's request that the statements made and the methods used be the results of my own experience.

First, the aftercare in cases of petrositis operated by following all chains of cells forward into the petrous pyramid until either a blind alley or pus is found. Sometimes more than one area is found from which pus escapes. The principle is the same in either case. At the time of operation the wound is left open. Gauze packing is used and this is so placed that the maximum amount of space is available for aftercare. A small rubber tube or wick of rubber tissue is placed near the discharging sinus. The gauze is packed tightly around the periphery of the wound but loosely around the drainage material. The great problem is to keep the small pathway clear, through which the pus is draining. Granulations quickly form and obstruct the drainage. In order to obtain free drainage for the number of weeks required for resolution, it is necessary to frequently remove these granulations. A ring curette or, on occasion, a small Spratt curette is necessary to remove both granulations and accumulated detritus. Occasionally it is necessary to probe or curette the passageway. A small suction tube is a handy aid and promotes drainage. The surprising feature of these cases is the facility with which resolution occurs when the small size of the canal for drainage is considered. After all symptoms have disappeared and drainage has ceased, a secondary plastic operation produces good cosmetic results. If a radical mastoid operation has been necessary the "flap" is made at the time of the plastic procedure. I believe the skin graft operations are contraindicated because of the theoretical possibility that the epithelium might seal off a partially resolved infection.

The eventual healing time, of course, varies considerably.

The postoperative treatment of a patient who has had a petrous exposure by the gasserian ganglion approach offers an entirely different problem in aftercare. Sometimes these patients will have had a previously performed "searching" operation. If the gasserian ganglion or direct approach to the petrous is then done, drainage may be established through the first excavation, and the second incision may be closed. If the direct approach is used as a primary operation, then the drainage may be accomplished through the operative incision. Complete resolution is hastened by this direct and adequate drainage. Rubber tissue or oiled silk make satisfactory wick drains. Both may be reinserted without damage and without pain. Drains are, of course, removed when the discharge ceases. The resulting scar is not noticeable, most of its course being beyond the hair line. One precaution should always be observed. Some retaining appliance should transfix the drain and remain outside the skin. Drains may be easily lost and are most difficult to recover. These drains may also adhere to the dressing, and unless the dressing is inspected by the doctor, he may be chagrined to learn that the search for a lost drain should have been in the waste bucket instead of the patient's head. Large safety pins make good identifying instruments.

Some generalizations regarding postoperative wounds will form my conclusion.

One broad statement which will, of course, be challenged, is "leave the wound alone." This statement is necessitated by the punching and probing, wiping and rubbing, pulling and hauling which is all too common. It is painful to the patient, and I am now convinced delays the natural, prompt healing.

Medicines and medicated dressings are also used in extremes. Each doctor has his own pet solutions and mine are limited to saline or a combination of boric and alcohol, equal parts. Because of a long unbroken habit, I had used iodoform gauze as the operative dressing of choice. I have discontinued this practice, and, after flushing the wound with alcohol, I cease medications except the cleansing normal saline. The common practice of wiping the inside of a wound with cotton and medications I have discontinued. I believe it disturbs the delicate growth of both granulations and epithelium. Other medications which have, on occasion, proved their usefulness, are balsam of peru to stimulate sluggish wounds and silver nitrate cautery applied to little tongues of excess granulation which prevent

apposition of the growing epithelial wound margins. It is sometimes necessary to clip these tongues of granulations with scissors. The procedure is painless.

Bandaging is discontinued as soon as possible, and the gratitude of the patient on being rid of this cumbersome accoutrement more than compensates for the potential advertising value. Adhesive plaster is irritating to some skins, but if properly handled gives little trouble. Removing adhesive may be accomplished without discomfort. Mopping the surface with a cotton ball soaked in gasoline and then allowing a few moments to elapse will permit the removal without pulling the hair or denuding the skin. Some of the adhesive material may remain on the skin and this can be removed in the same manner.

Surgery in otology was developed so efficiently by the pioneers that we all remember that little has been added by our own generation. Perhaps surgery of the petrous process is the one notable exception. As I look back over the past twenty years and recall the painful dressings, the rather frequent poor cosmetic results, the long convalescence and the reduced function, it is with the thought that there has been a gradual but unheralded improvement in most of these items. Perhaps the value of a paper on postoperative care lies not so much in the citation of procedures which are accepted by most of you, as in refreshing our efforts for still further improvement in these commonplace surgical chores.

121 East 60th St.

XLVIII

CARE OF MASTOID WOUNDS

FOLLOWING THE RADICAL AND MODIFIED RADICAL MASTOID OPERATIONS*

J. Morrisset Smith, M.D.

New York

Securing satisfactory results from the radical mastoid operation constitutes one of the major problems in the work of the average otologist, first because of the complex nature of the operation and next because of the few opportunities the average individual has to perform it.

In many instances the discharge not only refuses to stop but the foul odor persists, accompanied by an excessive production of infected granulation tissue. The operator in his efforts to correct these faults tries a wide variety of methods in the after-care with very little success.

There is little doubt that many of these cases are over-treated. Most of the unnecessary after-treatment is primarily due to the failure of the operator to adequately remove the diseased bone and granulation tissue at the time of the operation, so that even the most skillful after-treatment fails to obtain the desired results.

It is therefore impossible to help with these problems without dealing with some of the major steps so necessarys in the operative technique.

ERRORS IN SURGICAL TECHNIQUE

The chief fault with the average operator is the failure to properly lower the facial ridge. This is best accomplished by using a sharp Richards curette to gently shave away the bone over the course of the mastoid segment of the facial nerve from the horizontal semicircular canal down to a line parallel to the level of the floor of the middle ear. It is especially necessary to reduce the

*Presented as part of a Symposium on Care of Mastoid Wounds before the Annual Meeting of the American Laryngological, Rhinological and Otological Society, Rye, N. Y., May, 1939.

bone over the lower part of the mastoid segment next to the floor of the tympanum.

This creates a shallow cavity from which the granulations are easily removed and eliminates the deep pocket so frequently left in the floor of the tympanic cavity. The vertical portion of the facial ridge overhanging the region of the oval and round window is removed in the same manner. The facial ridge may be safely lowered by this method, since a warning twitch will always precede a serious injury to the nerve.

All of the diseased bone in the mastoid should be removed to healthy plate both in the middle and posterior fossæ, the dense bone forming the external wall of the attic must be removed to the thin dural plate forming the floor of the middle fossa. This allows free access to the roof of the cavity. The annulus is completely removed along with the granulations in the eustachian tube and middle ear; then the cells in the region of the tube and the hypotympanic space are exenterated.

With these major steps of the operation properly executed, there is a shallow cavity free from granulations and bony pockets with smooth walls ready for closure and dressing.

CUTTING THE FLAP

It is very important to secure a satisfactory flap. The technique described in the writer's original article on the radical mastoid operation* is as follows: The flap is always cut posteriorly in the concha, in order to secure a uniform opening of the correct size, its limits should be outlined on the skin in the concha with the point of the flap knife. The outline starts at the bottom of the membranous canal, making a half-circle backwards and upwards to a point from one-half to three-fourths of an inch in the concha, then upwards and forward to the roof of the membranous canal. The knife is then inserted through the concha at the outermost part of the half-circle into the mastoid cavity, the operator watching for the point of exit of the knife behind to see that the proper amount of postauricular tissue is included in the flap. The incision follows the half-circle outlined upward to the roof and stops, the knife is then reversed and follows the lower half of the outline to the floor of the canal; the flap is then divided in the center. The cartilage and soft tissues are dissected away from both halves of the flap,

*Transactions of the American Otological Society, May, 1930.

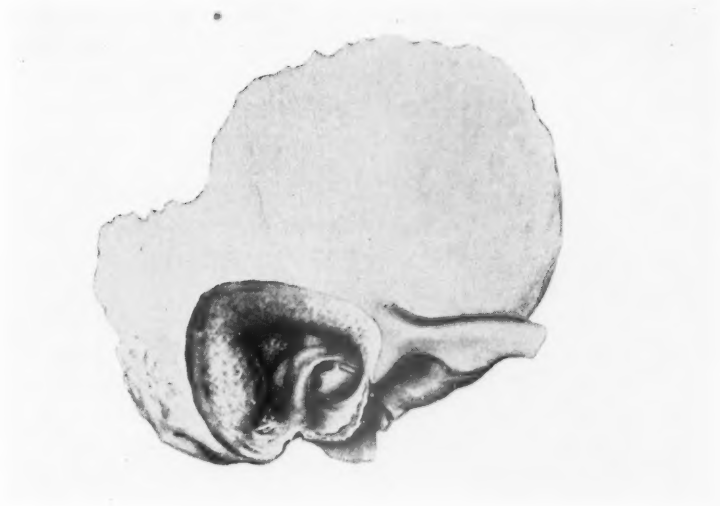

Fig. 1. Right radical mastoid operation.

permitting the skin from the upper half to be placed against the roof and the lower half against the floor.

CLOSURE OF THE WOUND

The tension on the external ear is relieved by removing the retractors and particular care taken to control all bleeding by pressure and ligation.

The wound is thoroughly cleansed with peroxide and salt solution and completely dried. It is then lined with strips of rubber tissue, the type used in making cigarette drains, and packed with iodoform gauze. The wound must be entirely free from fresh or clotted blood when this is done. The periosteum and temporal fascia are sewed back in their original position with interrupted catgut sutures. This serves two purposes: it prevents postoperative drooping of the ear and also provides suitable access to the roof of the cavity. The entire skin incision is closed with skin clips and an external dressing applied. If dura or sinus are exposed, a small piece of idoform gauze is placed next to the exposure and the rest of the cavity lined with rubber tissue. The advantages of the rubber tissue are threefold. It avoids the very painful first dressing so bitterly complained of by the patient. The rubber tissue pre-

vents the gauze from adhering so that both may be removed without pain; finally the rubber tissue avoids the new granulations which become entwined in the meshes of the gauze when it is placed in the bare bone, this in turn minimizes the bleeding. Occasionally a plastic exudate may form between the bone and rubber tissue, if so it may be removed at the first dressing. If rubber tissue is not used, I prefer one half-inch albolene gauze.

THE FIRST DRESSING

It is especially important at this point to emphasize the necessity of beginning the after-treatment with a cavity free from new or clotted blood. The packing and rubber tissue are removed the second day, and if the bleeding is fully controlled, the packing is not replaced. Where the bleeding persists a pledget of 10 per cent cocain is used and a small piece of plain gauze moistened with 95 per cent alcohol is placed in the cavity for twenty-four hours, the packing discontinued as soon as the bleeding is controlled. I would prefer to omit the packing at the conclusion of the operation. However, the bottom of the cavity may become filled with blood, and fresh or clotted blood in the wound furnishes a good media for bacteria as well as an excellent base for the growth of new granulations. It is therefore necessary to have patience at the first two or three dressings and remove all blood until the bleeding is fully stopped. The external dressing is now permanently removed to allow thorough aeration of the cavity; a small dressing is placed over the mastoid incision. The skin over the mastoid incision and at the entrance to the external meatus is kept scrupulously clean with peroxide and alcohol. Irrigations are not used unless there is a secondary purulent infection present, then boric irrigations are used three times daily, followed by ten drops of 95 per cent alcohol. If the alcohol drops are too painful, a few drops of 10 per cent cocain solution used shortly before the dressings will relieve most of the pain. Alcohol is one of the best deterrants to unwanted granulations.

All unnecessary manipulation of the interior of the wound should be avoided. I employ silver nitrate very sparingly in these cases. It is especially useful in removing the superficial layer of infected granulations, but should not be placed on bare bone or used in strong solution over the region of the tympanic segment of the facial nerve. If excessive granulations appear, a pledget of 10 per cent cocain is used and the granulations removed with a sharp curette, avoiding the region of the stapes and the facial nerve as well as any areas of exposed sinus or dura. A temporary pack of

alcohol gauze may be used where it has been necessary to curette an extensive area of granulations.

It has been my experience that some of the radical wounds will fill in with healthy granulation tissue in spite of the most complete operation and careful after-treatment. Aside from the fact that it destroys any hearing that remains, I see no real objection to it. In a large majority of the cases, the radical operation is indicated to remove the danger to the life of the individual, with the hearing so markedly impaired that there is litle chance of improvement. However, where the hearing is of vital importance, a skin graft offers the best chance of keeping the internal wall of the middle ear free from granulation tissue, a factor so necessary for the conveyance of sounds to the inner ear.

Daily dressing with careful inspection of the wound by the operator is absolutely essential to secure the best results. Where it is impossible to check the growth of granulations, firm packing of alcohol gauze is used every day or second day to be sure that the wound fills in from the bottom with healthy tissue. As a rule, I follow the general outline given above, but in the final analysis the use of packing, drops and irrigations are best dictated by the progress in the individual case.

A slight mucoid discharge free from odor persisting after operation is usually from the tube or mucous membrane, and while annoying is not dangerous and requires no further operation. A foul smelling discharge persisting after a long period of careful after-treatment usually means a residual area of necrotic bone and usually requires a secondary operation.

The same meticulous daily care is necessary to obtain satisfactory results in the modified radical operation.

After the flap is cut and bleeding controlled, the packing is placed in the posterior part of the wound but should not rest on the remnants of the drum and ossicles. The skin in the portion of the external canal not involved in the operative procedure is carefully preserved. Plain gauze packing is used and every effort is made to keep the granulations away from the antrum. Where a secondary infection occurs, peroxide followed by gentle irrigations should be used to avoid disturbing the malleus and incus.

Dermatization of the antral space without granulation tissue is the desired objective.

123 East 53rd Street.

XLIX

CARE OF MASTOID WOUNDS

FOLLOWING OPERATIONS ON ABSCESS OF THE BRAIN*

HAROLD G. TOBEY, M.D.

BOSTON, MASS.

A consideration of the after-care of the mastoid wound in relation to brain abscess of otitic origin necessitates a very brief review of the characteristics of the various types of the lesion, because the type so often influences the site and the manner of treatment. The character and situation of the lesion, as well as the experience of the surgeon, will direct the procedure.

Otitic abscesses may be classified as acute and subacute or chronic, with perhaps only a two to four weeks' interval in progressing from the acute to the subacute stage. They are also differentiated into abscesses arising by direct extension from the petrous bone or by metastasis from the blood stream, the former since they originate by gradual extension from the petrous bone and by a retrograde thrombosis usually result in the chronic encapsulated abscess. The latter or the metastatic lesions result in the acute, nonencapsulated abscess, often superficial, accompanied by a varying amount of encephalitis surrounding the area. It is in this type that the higher mortality exists because of a greater tendency for invasion of the meninges early in the disease and not infrequently necessitates intervention before the time of any possible walling off of the infected area can be accomplished.

The dictates of surgery usually demand the drainage of pus wherever found, and this holds true for suppuration in the brain. However, a number of factors have to be taken into consideration, such as (1) time of drainage, (2) site of drainage and (3) method of drainage.

Time of Drainage: It is universally accepted that the ideal time for drainage of the abscess is to await the encapsulation. This

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usually occurs during the fourth or fifth week and at times seems to occur much earlier.

However, during the acute stage with high temperature, marked prostration, an increasing number of polymorphonuclear cells in the spinal fluid, and signs of a beginning meningitis, early intervention may be necessary.

Site of Drainage: The choice of the site of drainage presupposes that a complete and thorough mastoidectomy has been performed, either a simple or a radical operation, according to the condition present. If indications of intracranial involvement are present, a thorough exposure of the dura in the middle and posterior fossæ is necessary at the primary operation.

This exposure of the dura is yet more necessary during secondary mastoidectomy necessitated by the onset of symptoms of brain abscess with an unhealed mastoid wound.

It is not usual to find any evidence of direct extension from the mastoid process or the middle ear in cases of acute brain abscess; however, in the presence of a subacute or chronic abscess there is frequently found evidence of such a direct extension, such as caries of tegmen antri disclosing granulations upon a thickened dura and occasionally the presence of a distinct fistulous tract connecting with the abscess. Under such circumstances it is almost the universal custom among otologists to institute drainage in this area.

In acute abscess and in chronic abscess developing after the healing of the mastoid wound and middle ear, the point of election is through a clean area as nearly as possible directly over the site of the abscess, if there are adequate localizing symptoms.

Methods of Drainage: Methods of drainage are varied and numerous and will depend upon the previous experience of the surgeon. (1) MacEwen obtained good results in acute abscesses by simple evacuation through an exploring cannula, not allowing for any drainage whatever, but in the chronic encapsulated abscess he used a decalcified chicken bone; however, it is generally held at the present time that some form of drainage is necessary for the acute as well as for the chronic abscess.

(2) Dandy has advised tapping the abscess and repeating the procedure if the symptoms persist; (3) LeMaitre, a gradual dilata-

tion of a tract leading from the cortex to the abscess. (4) The technique of J. E. King consists of a wide exposure and unroofing of the abscess with a deliberate herniation which eventually subsides. This procedure is necessarily confined to treatment of cerebral abscesses on account of the wide exposure and is not applicable to cerebellar abscesses.

Between these two extremes is the introduction of some drainage material into the abscess. Gauze and rubber strips have been used to pack the cavity; glass and hard rubber tubes have been largely discarded; rubber tubing which does not collapse is frequently used. (5) At the Massachusetts Eye and Ear Infirmary the Mosher wire basket drain has been used for a number of years with marked success.

The introduction of the drain has in recent years been greatly facilitated by the use of the diathermy knife for the removal of the overlying cortex, particularly in deep-seated abscesses. This is accomplished by the use of the wire loop or the straight needle for the removal of a core of the cortical tissue down to and including a sufficient portion of the abscess capsule.

The removal of this tissue may be preceded by a coagulation of the dura over the exposed site, and if possible a delay of twenty-four to forty-eight hours before the actual insertion of the drain to allow for a more thorough walling off of the meninges.

Following this procedure there seems to have been much less postoperative herniation of brain tissue, which has so often been troublesome.

There is established, also, a wide tract to the abscess cavity which allows for a thorough exploration and facilitates the introduction of the drainage material. A rubber tubing of sufficient size can be easily introduced, and whatever objection there may be to the use of the wire basket drain, on account of its rigidity, is overcome by the ease with which it can be properly placed.

This technic is equally applicable to either cerebral or cerebellar abscesses and to direct drainage through the mastoid wound or through a clean area outside of the mastoid wound.

The drain should be sutured to the skin during the period of active suppuration; anchoring the drain to the dura is not necessary or advisable after the use of the diathermy.

Postoperative Care: Maintenance of adequate drainage is essential. Rubber tubing of sufficient calibre will afford inspection of the cavity under reflected light after a careful clearing of the tube, for which a very gentle suction is convenient.

The wire basket drain demands almost daily curettage of the inner surface for the removal of the granulations which spring up and project through the wire mesh, tending to interfere with drainage as well as inspection of the cavity; however, this characteristic aids in the retention of the drain in its proper position, which is so important.

The duration of drainage will vary with the individual patient, four weeks being considered the average time when the suture may be removed and the drain will be extruded by intracranial pressure or, in the case of the rubber tube, gradually shortened. The wire basket at times will be extruded, but more often it will be necessary to separate it from the surrounding granulations on the outer circumference. This is accomplished by a final thorough curettage of the inner surface of the drain, accompanied by a gentle rocking and twisting motion with the gradual withdrawal. Occasionally the granulations will have to be separated from the outside of the drain with some such instrument as a submucous elevator.

Irrigations of the abscess cavity are not carried out at the Massachusetts Eye and Ear Infirmary, and it is needless to say that if irrigations are deemed necessary, adequate provisions must be made for the return flow of the fluid. The dressings should be done daily, preferably by the surgeon himself, or at least under his immediate supervision.

Herniation of the brain may occasionally be a troublesome feature, but as King has shown, can be successfully overcome. At times the brain tissue may become strangulated and necrotic, in which case it can be removed by scissors or by the diathermy knife.

Complications: The uncomplicated case of otitic brain abscess successfully diagnosed and drained, proceeds to a cure progressively with a return to consciousness within a few hours and a slower but continuous subsidence of the localizing signs. Unfortunately, complications are prone to occur. The acute abscess which has necessitated early intervention may continue with a spreading encephalitis which terminates in a septic meningitis. The patient

with a chronic abscess which has apparently been successfully drained may continue with signs of pressure or, after a lapse of a few days, the pressure signs recur. Two possibilities present themselves, namely, an extension from the abscess which has been drained or the presence of multiple abscesses.

In case such a situation arises, and it often does, the abscess must be re-explored for a possible extension, using the encephaloscope or a long-bladed forcep. The instillation of iodized poppy seed oil is rarely successful in revealing an extension which is shut off from the original abscess and is filled with pus under pressure, but will reveal an extension which is open and draining. Digital exploration, which is generally frowned upon, has proved successful in several cases with ultimate recovery. The exploring finger can be easily introduced into the fistulous tract where there has been a sufficient removal of the dura by diathermy.

The success of the after-treatment in the presence of multiple abscesses will depend upon the ability of the surgeon to localize and drain them.

Combatting Intracranial Pressure: Intracranial pressure may be controlled in several ways; for instance, by lumbar puncture, ventricular puncture, by hypertonic solutions given either intravenously or rectally, and by decreasing the amount of fluid intake.

Ventricular puncture and hypertonic solutions given intravenously are rarely used at the Massachusetts Eye and Ear Infirmary. Ventricular puncture practically never is used, but occasional use is made of the hypertonic solutions during the period of awaiting the encapsulation. Lumbar puncture, however, has been found to be very efficacious in reducing the pressure, as well as in watching the progress of the lesion and as an aid in diagnosis.

Cahill has pointed out that thrombosis of the venous sinuses involving the torcular has been found to be the cause of a long-continued increased intracranial pressure, for which frequent lumbar punctures over a long period of time have afforded the only relief.

In the presence of a suspected cerebellar abscess, great care should be used in the initial stages, since the sudden removal of a large amount of spinal fluid may cause a prolapse of the medulla into the occipital foramen.

270 COMMONWEALTH AVENUE.

BIBLIOGRAPHY

1. Cahill, H. P.: Modern Treatment of Brain Abscesses. J. A. M. A., 102:273, 1934.

2. Dandy, W. E.: Treatment of Chronic Abscess of Brain by Tapping. J. A. M. A., 87:1477, 1926.

3. King, J. E. J.: Treatment of Brain Abscess. Laryngoscope, 34:974, 1924.

4. LeMaitre, F.: Walling Off of Subarachnoid Spaces, Etc. ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY, 29:1 (Mar.), 1920.

5. MacEwen, W.: Pyogenic Infectious Diseases of the Brain and Spinal Cord. J. Maclehose & Sons, Glasgow, 1893.

6. Mosher, H. P.: Wire Gauze Brain Drain. Trans. Am. Otolog. Soc., 14:102, 1916.

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A NOTE ON THE GREATER DISABILITY FOR HEARING HIGH TONES IN CASES OF CONDUCTION DEAFNESS*

A. G. POHLMAN, M.D.

LOS ANGELES

Some years ago Alexander[1] submitted the evidence in favor of the accepted idea of a dual conduction of air sounds to the cochlea. This interpretation assumes that the low tones are conducted through the ossicular chain, while the high tones pass into the cochlea through the round window membrane. The writer[2] reviewed the paper presented by Alexander, and held for the opposing viewpoint which postulates that the drum membrane and ossicular chain is the only effective route for the conduction of air vibrations of all frequencies. It was pointed out that the normal functional responses in the cochlea which are less sensitive to low tones, had been mistaken for a frequency limitation in the conduction apparatus. Further evidence for the validity of this interpretation has been presented in the effects obtained where the middle ear apparatus has been replaced by a mechanical prosthesis patterned after the sound conduction apparatus in birds.[3]

The recent work of Crowe and Guild[4] and the more comprehensive report by Crowe and Baylor[5] have attracted attention to a type of conduction deafness in which there is a greater disability for hearing high tones than for low tones. These investigators ascribe the condition to an obstruction of the auditory tube with lymphoid tissue and report the recovery on the removal of the obstructing tissue by means of radium treatment. They[5] also indicate that a beginning of a conduction deafness may be found in the lower sensitivity to high tones, and feel that a proper early treatment may result in a reduction in the incidence of conduction deafness by 50 per cent. If this latter conclusion is substantiated, the work of Crowe and his co-workers has developed one of the most important advances in modern otology.

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*From the Department of Otolaryngology, University of Southern California.

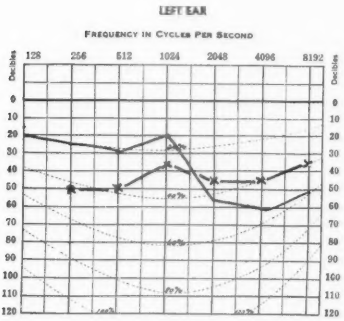


Fig. 1.

It will be noted that the diagnosis in these cases, reported from the Otological Research Laboratory of Johns Hopkins University, was established as a result of the treatment. It is unfortunate that these investigators did not report on the bone conduction sensitivity other than that elicited through a 512 fork test. The writer was interested in the possibility of establishing a diagnosis through a correlation between the air and the bone sensitivity, and also in the probability that the described disability might be due to causes other than an obstruction of the auditory tube. The condition is certainly not common in adults and only recently two cases came to my attention which are worth reporting briefly.

K., a young adult, came in for examination, complaining of lowered acuity with marked deformation in speech reception on the right. The left ear he felt was quite normal and this was confirmed. The right ear had been operated some years ago (simple mastoid) and required reoperation. The drum membrane was intact and somewhat thickened. The air acuity curve is shown as a solid line (Fig. 1) and would have been interpreted as characteristic of a mixed deafness with an incomplete conduction deafness component (Rinne positive) and a superimposed perception deafness for high frequencies—the usual perception deafness. However, on taking the bone sensitivity with the normal ear masked, the relatively normal curve, indicated in the broken line, was developed. Accordingly, the case was listed as one of conduction deafness and Rinne positive for low frequencies and Rinne negative for high frequencies.

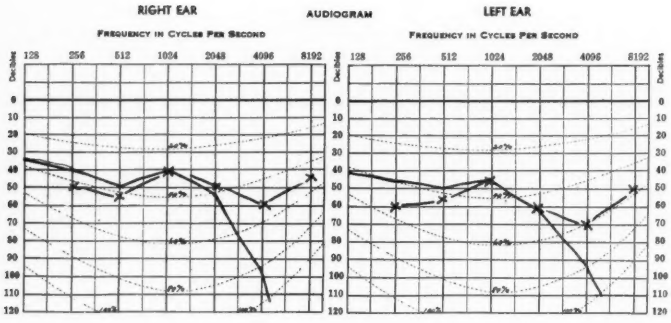


Fig. 2.

The bone sensitivity for high frequencies could not have been enhanced, because as Pohlman and Kranz first pointed out,[6] no prolonged bone conduction can be demonstrated above 2048. In this case the auditory tube was obviously patent.

B. gave the history of an otosclerosis and had been observed by a number of otologists who agreed in the diagnosis. The drum membranes appeared relatively normal; the Rinne was negative; the acoustic fan test was positive; and the acoustic probe against the malleus was negative. The diagnosis of an ankylosis of the stapes accordingly seemed to be quite definite. The air conduction curves for both ears are shown as solid lines in Fig. 2, and suggested an involvement of the otic capsule (perceptive deafness). However, the bone sensitivity (in broken line) indicated a normal internal ear on both sides. Here again we note a case of greater disability for the hearing of high frequency air sounds where the blocking is quite definitely at the stapes footplate.

These two cases are undoubtedly an exception to the general rule and are the first two of the kind that have come to my attention. They indicate, however, that this curious limitation in the conduction of high frequencies may be due to causes other than a blocking of the auditory tube. They also emphasize the importance of an audiometric examination throughout the frequency range before a definite diagnosis of a mixed deafness may be made.

2202 West Third Street.

BIBLIOGRAPHY

1. Alexander, G.: Die Theorie der Luftleitung mit Berucksichigung der Anatomie und Klinik. Monatsch. f. Ohrenheilk, 65:1-173, 1930.

2. Pohlman, A. G.: Conduction of Air Sounds to the Cochlea. ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY, 40:717, 1931.

3. Pohlman, A. G.: Mechanics of Middle Ear Prosthesis. Ztsch. f. Hals-Nasen-u- Ohrenheilk., 40:487, 1936.

4. Crowe, S. J., and Guild, S. R.: Impaired Hearing for High Tones. Acta Otolaryngologica, 26:138, 1938.

5. Crowe, S. J., and Baylor, J. W.: The Prevention of Deafness. J. A. M. A., 112:585, 1939.

6. Pohlman, A. G., and Kranz, F. W.: Quantitative Experimental Evidence on the Acuity for Hearing by Bone Transmission. Proc. Soc. Exp. Biol. and Med., 23:76, 1925.

LI

NASAL OBSTRUCTION CAUSED BY COLLAPSE OF THE NASAL ALAE*

HAROLD I. LILLIE, M.D.

AND

KINSEY M. SIMONTON, M.D.

ROCHESTER, MINNESOTA

Nasal obstruction is a frequent symptom which causes patients to seek relief. Actually, the degree of nasal obstruction in a given case may be difficult to estimate because many patients do not seem to know what to expect of the nose under normal conditions. The internal configuration of the nose may not be at fault. The method of breathing may cause the symptoms. Frequently patients are encountered who complain of inability to breathe through the nose merely because their own effort to breathe deeply causes a collapse of the alæ nasi on the inspiratory effort. They attempt to breathe "with the nose" instead of "through the nose." The desired effect of bellows action of the chest and abdomen is not utilized as it should be.

This type of nasal obstruction we choose to call "alar collapse."

Anatomic Considerations: According to Morris,[1] the nose is supported by a framework of bone and cartilage. The bony bridge is formed by the two nasal bones and the nasal processes of the frontal bones. The tip of the nose and the nasal vestibules are supported by a framework of cartilage. There are five principal nasal cartilages. The nasal septal cartilage in the median plane, the paired lateral nasal cartilages, and the greater alar cartilages, form the principal support. Smaller accessory nasal and sesamoid cartilages are present in the fibrous tissue of the nose, but they are not significant in relation to its contour or support. These cartilages are covered by firm fibro-adipose tissue and by the integument which completes the alæ of the nose.

The lateral nasal cartilages (Fig. 1) are flat, triangular expansions of the septal cartilage; the upper third portion of each nasal

*Section on Otolaryngology and Rhinology, The Mayo Clinic.

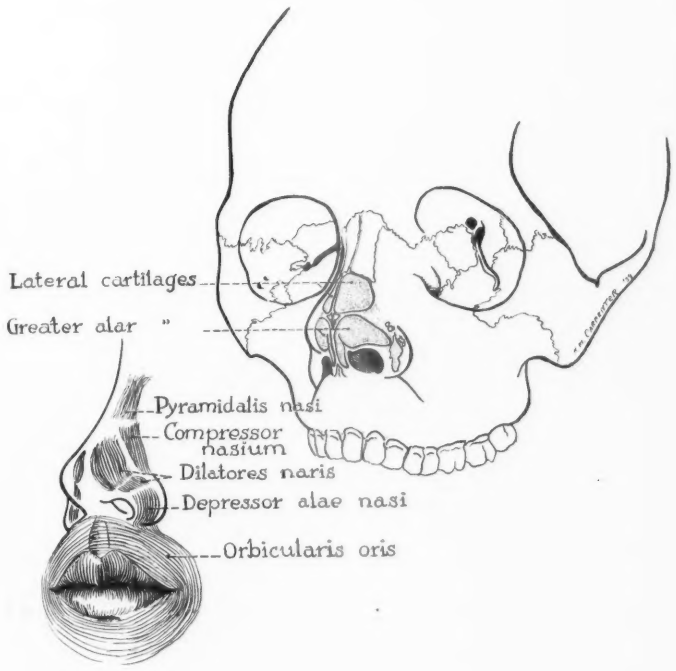


Fig. 1. Anatomic considerations of the cartilages and muscles of the nose, showing the structures affected in "alar collapse" and the muscles which affect these structures.

cartilage is continuous with the septal cartilage. They are firmly attached above to the nasal bones and to the nasal processes of the maxillæ. Below they are attached by fibrous tissues to the greater alar cartilages.

The greater alar cartilages are thin and are curved to form both a medial and a lateral crus. The medial crura are bound together to form the inferior part of the mobile septum. The point of curvature corresponds to the apex of the nose. The lateral crura curve dorsally. They are connected posteriorly to the nasal margins of the maxillæ by dense fibrous and fatty tissue. They help to maintain the contour of the nose. The greater alar, and to a lesser extent, the lateral nasal cartilages, are concerned in the production of alar collapse.

The cartilages of the alæ nasi are subject to a limited degree of motion by the action of the muscles of the nose. These muscles are of two kinds, the elevators and the dilators, which arise from above; and the depressors and contractors, which act from below (Fig. 1). The elevators consist of (1) the pars transversa of the nasalis muscle, which arises from the bridge of the nose and is inserted in the nasolabial sulcus, and (2) the caput angulare of the quadratus labii superioris muscle, which arises at the root of the nose and sends a fasciculus to the wing of the nose. The dilators are two in number. The dilatator naris posterior is a thin, triangular muscle which arises from the skin at the side of the nose and is inserted in the lower margin of the ala. The dilatator naris anterior is a small, usually poorly marked muscle, which arises from the lower margin of the cartilage at the front of the ala and is inserted in the skin near the margin of the nostrils. The depressors and contractors are (1) the pars alaris of the nasalis muscle, which is contractile and arises from the alveolar juga of the upper incisor and canine teeth and is inserted in the dorsal margin of the ala, and (2) the depressor septi nasi. Acting in various combinations, these muscles bring about a variety of results. The pars transversus of the nasalis and the caput angulare of the quadratus labii superioris, acting in unison, draw the wings laterally and upward, giving the expression sensuality. The pars transversus and the pars alaris, acting together, may constrict the nostrils. The pars alaris and the depressor septi draw the nostril downward.

Mechanism: Kyle[2] gives four causes of obstruction of the nasal vestibules: (1) faulty formation of the lateral cartilages; (2) lack of development caused by a blocked nose in childhood; (3) disuse atrophy of the dilator muscles in cases with chronic obstruction from other causes, and (4) "from the contour of a long, pointed nose with a long, pointed slit-like orifice, there may be a tucking-in and narrowing of the nasal orifice, due to the action of the constrictor muscles." To these causes we would add the effect of the excess action of the dilator muscles.

In our experience, Kyle's fourth hypothesis has been a frequent cause of subjective nasal obstruction. Persons having a long, narrow nasal orifice are particularly susceptible to alar collapse. Many such persons breathe freely during quiet respiration, but on forced inspiration a definite medial motion of the alæ nasi with subsequent narrowing of the nasal orifice, may be noted. The alar motion affects chiefly the lateral crus of the greater alar cartilage, which may be drawn inward throughout its extent by the preponderance

of the constrictor muscles. In certain instances, the greater alar cartilage is rotated on a horizontal axis so that its upper margin and the lower limits of the lateral nasal cartilage are turned mediad by the action of the dilator muscles at their insertion in the inferior borders of the greater alar cartilage. The motion of the alæ is more marked in elderly individuals who have a reduced elasticity of the tissues in whom the negative pressure of inspiration tends to close the vestibule. This alar motion is best observed when the patient is requested to breathe deeply.

Diagnosis: The diagnosis of nasal obstruction should include, first, a statement of the amount of subjective obstruction, and second, an objective estimation of the degree of obstruction. In estimating the actual degree of nasal obstruction in a given patient, several factors must be considered. Wright and Smith[3] have said, "The internal configuration of every nose, even of those we would pronounce normal, varies so greatly that every nasal chamber is a law to itself. Anterior and posterior rhinoscopy are often incapable of furnishing us with trustworthy information as to the efficiency of the nasal chambers in the performance of these functions. The statements of the patients are still more untrustworthy. Some fail to appreciate even extreme grades of nasal obstruction. Others complain of it when manifestly it does not exist. The clinical experience, the common sense of the physician, and his ability to judge of the patient's temperament are more important guides to the appreciation of how these functions are in reality being performed than the help his technical skill or the instruments of precision at his disposal furnish him."

According to one of us (Lillie[1]), "It can be observed clinically that nasal respiration is an adaptive reflex mechanism, lessening resistance when respiratory need is increased, and vice versa. Dilatation of the vessels, when it is not carried to the point of rendering insufficient the amount of air supplied to the lungs, renders the air, when it reaches the pharynx, not only warmer, more moist, and more free of dust and bacteria, but by filling the unnecessary space in the respiratory region of the nose, it directs a more copious supply of air toward the olfactory region."

The type of patient encountered is of first importance. Is he a chronic complainer, is he too introspective about little changes; does he understand the function of nasal respiration and how it is accomplished? The physical environment to which the patient is subjected; climate, home and working conditions must be consid-

ered. A variable climate, with frequent and marked changes in the weather predisposes to hypertrophy of the membrane of the upper portion of the respiratory tract, whereas such changes are not produced by a warm, equable climate. The temperature, humidity and presence of drafts in the home are significant. Variable temperatures and exposure to drafts produce frequent changes in vasomotor tone which result in swelling of the nasal membranes. The absolute humidity of cold air is low, even though the relative humidity may be near the saturation point. Warming the air without adding moisture to it lowers the relative humidity. In this way, the air of many homes becomes excessively dry during cold weather. This condition causes congestion of the nasal membranes, and drying of the mucous blanket of the nose, in some instances to the point of crust formation. It produces the sensation of a stuffy nose. Patients having marked atrophy of the nasal mucous membrane often complain of obstruction even when it does not exist.

Working conditions to which a patient may be subjected, such as the presence of dust, irritating gases, low humidity, extremes of temperature, and exposure to frequent changes in temperature affect the physiologic response of the interior of the nose. Hypertrophy of the nasal membrane accompanied by obstruction, is a common finding in the case of gasoline station attendants who go from a heated building into the cold air many times daily. Laundry workers, exposed to hot, moist atmosphere, may have atrophy of the nasal membranes.

The differential diagnosis of nasal obstruction should include a consideration of the physiologic, anatomic and pathologic factors.

Physiologic Factors: Heetderks[5] observed a definite cycle of activity of the nasal mucous membrane in 80 per cent of apparently normal individuals. While the membranes over the turbinate processes on one side were actively secreting mucus and shrinking, those on the other side were dry and became swollen. Due to passive congestion, the swelling was more marked on the dependent side with the individual in the recumbent position. Nasal obstruction was induced by a hot, dry atmosphere, whereas cold air caused an increase in secretion. The responses were more marked in adolescents than in older individuals.

The nasogenital relationship is often encountered as a congestion of the nasal membranes in newly married individuals.

Anatomic Factors: Anatomic causes of nasal obstruction embrace septal deviation or thickening arising from developmental

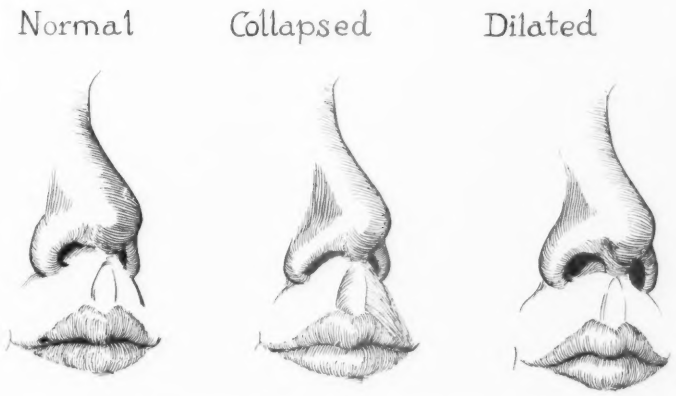


Fig. 2. Gross appearance of the ala of a normal nose; of a collapsed ala; and of a dilated ala.

anomalies, or from trauma, variations in the size of the nasal chambers and congenital occlusions of the nasal choanæ. The degree of obstruction arising from these causes will be determined by inspection of the nasal fossæ, aided by adequate shrinking of the membranes and use of the probe.

Pathologic Factors: Pathologic factors in the production of nasal obstruction are (1) infections which induce swelling of the nasal membranes, and in chronic stages, produce fibrosis and hypertrophy; (2) edema, hyperplasia, and formation of polyps associated with allergic disease; (3) simple hypertrophies resulting from long-continued physical irritation as previously mentioned; (4) swelling of the nasal membranes associated with metabolic disturbances, notably hypofunction of the thyroid gland; (5) edema caused by excessive use of vasoconstricting agents or watery solutions; (6) marked atrophy with crust formation, and (7) alar collapse.

Alar collapse may be found to be the sole cause of nasal obstruction, or it may be associated with one or more other anomalies. Frequently it is not appreciated, because of failure of the examiner to look for it in his haste to proceed with examination of the interior of the nose. The method of examination is simple inspection of the external nose during respiration (Fig. 2). It will be noted that the patient breathes "with the nose, not through the nose"; that inspirations are short and quick; and that the ala of the nose rotates

on a horizontal axis, the inferior margin turning lateral with a definite medial motion at the level of the superior margin of the greater alar cartilage, or the entire ala is drawn mediad. The efforts of the patient to draw air into the nose increase the degree of medial motion, while long, slow inspirations diminish the tendency to collapse. If these observations do not establish the diagnosis, a cotton ball tucked into the anterior superior recess of the vestibule will produce complete relief in uncomplicated instances of alar collapse. After the diagnosis of alar collapse has been made, the usual procedures will determine whether or not it is the sole cause of obstruction.

Treatment: When the examiner has satisfied himself that the nasal obstruction complained of is actually caused by alar collapse, the fact can be demonstrated to the patient; he can be requested to use his method of breathing before a mirror, so that he can actually see the effect. In turn, he can be shown how to use the dilating muscles of the alæ, so that he can see the difference in effect which they produce. This demonstration, in many instances, is all that will be necessary.

If collapse of the alæ cannot be overcome in such a simple manner, institution of other measures will be necessary.

A cotton ball of appropriate size may be tucked well forward into the anterior superior recess of the nasal vestibule, or a silver tube shaped to fit the vestibule may be used as suggested by Kyle. Such devices are available on the market. Thomson[6] feels that the collapse is the result of loss of muscle tone secondary to obstruction from other causes. He advocates removal of the primary obstruction and institution of breathing exercises performed before a mirror to restore muscle tone.

Several surgical procedures have been described to hold the vestibule open. Walsham[7] advocates rolling a strip of septal mucous membrane forward and suturing it in the anterior vestibular recess. Lack[8] turns a strip of the septal cartilage and membrane at right angles to hold the vestibule open. Brown[9] describes an operation designed to reduce the thickness of the columella nasi. An operation has been described in the performance of which a vertical wedge of tissue is removed from the medial aspect of the alar sulcus. Approximation of the edges of the incision bows the ala outward and diminishes its tendency to collapse. But surgical measures do not seem justified until the simpler measures have been given adequate trial.

SUMMARY AND CONCLUSIONS

A common, often unthought-of cause for the subjective sensation of nasal obstruction has been described as "alar collapse." It is more frequently encountered as a cause of nasal obstruction than is generally supposed. Suggestions for the relief of the symptom are made.

Collapse of the alæ nasi is a frequent cause of partial nasal obstruction, especially in persons having narrow noses. It is produced by overactivity of either the constrictor or dilator muscles of the nose and is influenced by the negative pressure of inspiration. The condition is often overlooked because of failure to look for it. Relief from symptoms may be obtained by a change in breathing habits, by wearing a device to hold the alæ open, or by a well directed plastic operation.

REFERENCES

1. Morris, Henry: Human Anatomy: A Complete Systematic Treatise. P. Blakiston's Son & Co., Philadelphia, edition 9, pp. 1296-1298, 1933.

2. Kyle, D. B.: A Textbook of Diseases of the Nose and Throat. W. B. Saunders Company, Philadelphia, p. 312, 1907.

3. Wright, Jonathan, and Smith, Harmon: A Textbook of the Diseases of the Nose and Throat. Lea and Febiger, Philadelphia, p. 95, 1914.

4. Lillie, H. I.: Practical Physiology of the Nose. Minnesota Med., 21:621-628 (Sept.), 1938.

5. Heetderks, D. R.: Observations on the Reaction of Normal Nasal Mucous Membrane. Am. J. M. Sc., 174:231-244 (Aug.), 1927.

6. Thomson, St. Clair: Diseases of the Nose and Throat. D. Appleton and Company, New York, edition 3, pp. 125 and 182, 1926.

7. Walsham (quoted by Ballenger, W. L.): Diseases of the Nose, Throat and Ear, Medical and Surgical. Lea and Febiger, Philadelphia, pp. 307-308, 1925.

8. Lack (quoted by Ballenger, W. L.): Diseases of the Nose, Throat and Ear, Medical and Surgical. Lea and Febiger, Philadelphia, pp. 307-308, 1925.

9. Brown, Edgar: A Note on the Treatment of Alar Collapse. J. Laryng. & Otol., 46:545-546 (Aug.), 1931.

LII

EIGHTH NERVE HIGH TONE DEAFNESS FROM A NUTRITIONAL STANDPOINT*

(A FURTHER CONTRIBUTION)

GRANT SELFRIDGE, M.D.

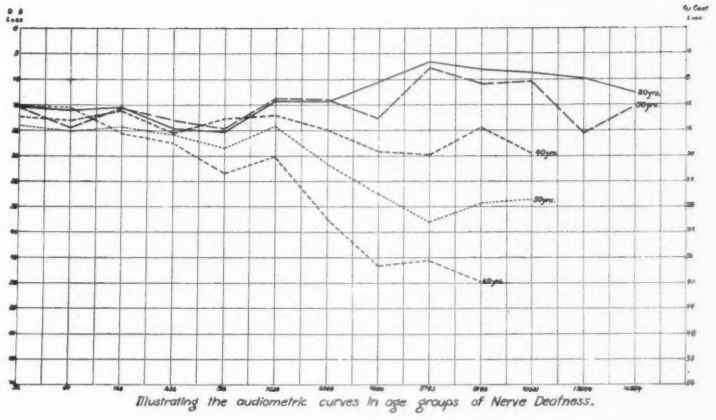
SAN FRANCISCO

One of the most distinguished members of the American Otological Society in the Harvey Lectures for 1931-32, in an address entitled "Underlying Causes of Deafness,"[1] speaking of the etiology, has this to say: "The ear, like the eye, is a highly specialized sense organ and is probably affected by general systemic disorders * * * " and further says: "As regard the role of vitamins and dietary insufficiencies and the use of salicylates and other drugs in the etiology of deafness, it is possible to learn much by feeding animals special diets, testing the transmission of sound * * *, and finally by making serial sections of the middle and inner ear of these animals for histological studies."

The same author, in a later article discussing the "Diagnosis and Differential Diagnosis of Deafness,"[2] calls attention "to changes brought about by age" and that "with each decade above the age of twenty there is increasing impairment for the higher frequencies." Further on he states, "When the audiogram shows good hearing for low tones but increasing impairment for each octave toward the high end of the scale, the lesion is an atrophy of the nerve supplying the basal turn of the cochlea. The cause is unknown."

He also says, "In the early stages of obstruction of the eustachian tubes in children the hearing is more impaired for high than for low tones." No explanation is given as to why in eustachian tube obstruction of children the high end of the tone scale is involved. Whether he believes as Gray,[3] that the nerve involvement of otosclerotics is primary and the bone changes are secondary, and that the bone changes are due to some factor disturbing the vasomotor arc and thus interfering with the nutrition of the nerve, was not stated. In a series of forty cases of conduction deafness to

*From the Harriman Research Laboratories of the Southern Pacific General Hospital.



Illustrating the audiometric curves in age groups of Nerve Deafness.

Fig. 1.

whom estrogenic substances have been given, all showed in their audiograms a drop in the high tone part of the scale. Most otologists of the past have looked upon the nerve changes as secondary to some infection, and yet recently Shattuck,[4] mentioning the views of several authors, says: "Vitamin deficiency should now be considered as a factor of possible importance in the mechanism of production of a great many cases of degeneration of the peripheral nerves, with or without evidence of cord lesions, which would have been attributed in former years to various other causes." It is hardly conceivable that all the cranial nerves participate in the degenerative processes in certain dietary deficiencies and that the eighth nerve should be exempt.

Just prior to the Harvey lectures, Nina Simmonds (co-author with Professor E. V. McCollum of "Newer Knowledge of Nutrition"), undertook a nutritional survey of a small group of cases of chronic progressive deafness, allergy and sinus disease, and as a result of this study a search of the literature followed to determine if there was sufficient evidence that certain general diseases had an underlying nutritional deficiency as a causative factor, and if so, that such evidence could furnish a basis for a claim that certain diseases of the ear, nose and throat could have a similar basis, especially as regards the eighth nerve. These studies were presented in an article entitled "Can Certain Diseases of the Ear, Nose and Throat, Especially Degeneration of the Eighth Nerve, Be Classified as Deficiency Diseases?"[5]

Further studies from the standpoint of nutritional deficiencies have been set forth in the following papers: "Eighth Nerve High Tone Deafness from a Nutritional Standpoint: A Preliminary Report";[6] "Chronic Progressive Deafness from a Nutritional Standpoint: A Preliminary Report,"[7] and "Nicotinic Acid and the Eighth Nerve: A Preliminary Report"[8] (in press). In addition to these papers is the article of Covell and Noble[9] in which is described in part their preliminary histopathological studies of animals and chicks deficient in B_1, B_{2}, B_6 and Jukes' chick dermatitis factor. The black tongue dog factor (pellagra) studies have not yet been completed.

The findings of Covell for the peripheral auditory mechanism of these dietary deficient animals should prove of interest. It is realized that while experimental studies aid in confirming or disproving results of this nature, and while it must be borne in mind that there is a difference between the experimentally induced deficiency and the milder type of deficiency in clinical cases, the evidence already offered by myself seems to be of enough value to warrant further study by interested otologists to either prove or disprove the findings already submitted.

Thus the study suggested by the author in his Harvey Lecture has been carried out with the exception of the animal hearing tests, and while more animal studies may be necessary, a sufficient number have already been studied to give positive evidence that the changes found are related to the deficient factors. The reason these hearing tests have not been made is due to the fact that no apparatus has been available for use in animals of such small size (rats).

AGE INCIDENCE IN RELATION TO HIGH TONE LOSS

It is granted that the eighth nerve is a peripheral nerve and the cochlea is the end-organ and that it is subject to the same degenerative changes as are found in all the various diseases and toxic conditions related to neuritis, as set forth in Spies'[10] book on "Vitamin B_1" and also in Shattuck's[4] recent article on "Nutritional Deficiency and the Nervous System."

It appears to me that the problem regarding the age incidence revolves about the question as to how early the signs of beginning degenerative diseases may be recognized and to what extent could one rely on early loss of high tones as seen in audiograms as confirmatory evidence. Also, to what extent these conditions are influenced by nutritional deficiencies.

The youngest case to show impaired hearing for high tones is a female child, aged eight years (to be reported later). The next youngest is a young woman of nineteen years of age (also to be reported later). Three cases, one aged twenty-two, one aged thirty-six and one aged thirty-nine, have already been reported on (see article on "Eighth Nerve"). These three cases showed definite dietary errors and the correction of these dietary errors, plus the addition of the vitamin B complex, brought the hearing back to normal. The case aged thirty-nine years has been seen recently and was found to have a relapse in hearing. A similar relapse has occurred in other and older cases but are showing definite improvement under thiamin and nicotinic acid. It is quite possible that in the future if the parenteral use of thiamin chloride does not stimulate the intestine to pick out a sufficient amount of the B supplied by a normal diet, that some preparation like the rice bran extract, liver or yeast, containing all the factors of the B complex, may be necessary for several periods during each year.

The question now arises, what part do lack of vitamins play in the degeneration of the nerve and what other factors contribute to it? For the past several months, laboratory methods for the study of the urine output for the B_1 and porphyrin, as well as the blood for vitamin C, have been instituted not only for nerve deafness but also for conduction deafness. In all, eighty-one cases have been studied: forty-seven nerve deafness cases, and thirty-four conduction deafness cases. The B_1 output has been low in all. In several the output has been 6 I.U., the lowest being 4 I.U. (normal is 20 to 35 I.U.). Porphyrin has been increased in approximately 50 per cent of the cases. Porphyrin appears to be an index for the use of nicotinic acid, although I have used it in all cases of hearing loss of high tones whether diagnosed as nerve or conduction plus perception deafness.

Present-day literature abounds with articles dealing with nutritional factors and disease, and the evidence appears conclusive that the neurological changes in pernicious anemia, beri-beri and pellagra are caused by dietary deficiencies.

Charles French,[11] in discussing the neuromuscular manifestations of beri-beri in experimental rats, says: "These results indicate an early involvement in beri-beri of the vestibular system of the nerve pathways serving vestibular nystagmus." He also says elsewhere: "The work of Peters and his group on the oxygen uptake of brain tissue indicates a disturbance of tissue metabolism in B_1 deficiency and offers a possible explanation for the progressive changes

in vestibular functions, as well as for tissue changes and hemorrhages in the brain system and cerebellum which occur later in the deficiency." With these findings it is most difficult to believe that the auditory nerve and cochlea can escape damage.

The recent articles of Shattuck, "Nutritional Deficiency and the Nervous System"[4] and "Landry's Paralysis in Relation to Vitamin B Deficiency,"[12] furnish much food for thought, as do also Lewy's[13] reference to alcoholic neuropathy and Jolliffe's and Goodhart's "Effects of B_1 Therapy on the Polyneuritis of Alcoholic Addicts."[14]

OTHER VITAMINS

Mellanby[15] has dealt thoroughly with vitamin A. Ono's[16] studies were carried on with animals chronically deficient in vitamin C and showed definite changes in the nerve. Further studies in chronically C deficient animals may confirm Ono's findings. Covell[17] has studied A, B, C and D. His studies with C were with acute deficiency and showed only slight changes in the nerve. "Meyer and McCormick describe degenerative changes in the peripheral nerves and the large cells of the anterior horns of the spinal cord in 'Studies in Scurvy'."[18] Studies are now in progress with vitamins D_2 and D_3. The position of some of the electrolytes has been studied (magnesium[19] and calcium[20]), the histological studies of which are to be undertaken.

If the lack of some particular fractions of the vitamin B complex (the most obvious deficiency in the human dietary today) can explain the changes in the nerve and vasomotor mechanism, further information is needed about the relationship of the electrolytes and the control of the body metabolic processes by the endocrine gland system, their actions, reactions and interactions. Williams and Spies[21] say: "It is becoming increasingly clear that an improper balance between the active principles derived from the glands of internal secretion and the vitamins and other essential substances contained in food can lead to various diseases. These materials from the glands and from the foods must eventually form a suitable environment for the cells of the tissue * * *. Its absence (B_1), however, results in cellular alteration in the nervous system, intestine, pancreas, stomach, salivary glands, liver, adrenals, and gonads, and probably in other tissues." It is quite possible that if there is an interrelation between vitamin B and the endocrines, especially the pituitary in relation to water balance, and the adrenal cortex as the stabilizer of electrolytes, especially of sodium, postas-

sium and magnesium, then it offers an explanation of the Ménière's syndrome. I have seen several instances of relief of mild dizzy spells following the parenteral use of B_1.

I also saw in consultation some years ago a pituitary case who had symptoms of Ménière's disease with an eustachian tube so blocked up that I was able only on one occasion after the use of a small bougie to get any air through the tube. This patient's fluid intake was restricted to eight ounces daily, and given some pituitary extract. In a short period the tube became normal and she was relieved of her vertigo. Apparently her disturbance was related to disturbances of water balance.

The chief function of vitamin B is to regulate the most fundamental of all life processes, cell respiration, and in view of its role in the conversion of carbohydrates to pyruvic acid and lactic acid as demonstrated by Peters, the evidence herewith submitted seems almost conclusive. If and when pooled samples of the endolymph in black tongue dogs can be examined for lactic acid by microchemical methods and are found to be positive, then the destruction of Corti's organ in the lower whorl of the cochlea is subject to definite explanation and related probably to nutritional deficiencies.

These authors, Williams and Spies, quoting Evans and others, state that male animals deficient in B_1 lose their libido—humans also—and in female animals there is a suppression of follecular function. Its lack also influences reproduction, pregnancy and lactation.

Thus it seems evident that as age advances and sex activity becomes lessened, degenerative changes become more evident after the age of fifty and may play an important role in so far as presbycusia is concerned.

BODY REQUIREMENT FOR VITAMIN B_1

Cowgill[22] says: "The minimum daily vitamin B_1 intake necessary for the adult human being weighing ninety-nine pounds is about 135 I.U.; one weighing 154 pounds needs approximately 280 I.U., and one weighing 198 pounds needs about 500 I.U."

Jolliffe and Goodhart[11] found in the treatment of neuritis in alcoholic addicts that "the minimum effective dose was of the order of 4 mg. daily (1200 I.U. of thiamin chloride); from 10 to 20 mg. (3000 to 6000 I.U.) gave more valuable results; and some cases

as much as 50 mg (15,000 I.U.) had to be given to secure improvement." Weiss,[23] in the treatment of his heart cases, has made similar observations.

In our early use of B_1 parenterally, the subject of dosage for patients of different decades was discussed with Mrs. Emerson of Herbert Evans' Department of Biological Research, who said that they had found the requirements for older animals were greater than in young ones. With this information it was decided to use 10 mg. (3000 I.U. per cc.) six days per week over a period of several weeks, depending on the age of the patient and the amount of B_1 present in the twenty-four hour urine specimen in order to secure a more rapid saturation of the body cells.

As the urinary output improves, the dosage can be reduced. In many instances similar results can no doubt be obtained by giving the thiamin chloride by mouth, especially in the younger age group in doses of 4 mg. night and morning.

TOXICITY

Chauncey Leake reports that at the recent meeting of Physiologists at Zurich, that no toxic symptoms occurred until massive doses of 10 grams were given with curare-like convulsions. Steinberg[24] reports three cases of herpes zoster following administration of large doses of vitamin B_1 (1000 I.U.). He concluded that "the above case histories would seem to indicate that large doses of vitamin B_1 are capable of irritating the peripheral nerve plates." Apparently this toxic symptom occurs in only a very small percentage of cases treated with massive doses of vitamin B_1. In this series of cases it was less than one per cent. However, he says that one should be on guard in the use of this substance, and when an individual so treated begins to complain of intense burning pain in an unsuspected area, the administration of this vitamin should be stopped.

NICOTINIC ACID

Vilter, Spies and Mathews[25] say, "Since there is no evidence that pyridine can be formed in the human body, it is evident that nicotinic acid or its amide must be an important article of human diet, and that it is apparently necessary for the normal health of the mucosa of the mouth and intestine, nervous system, and skin, since these all are impaired in pellagra and improved by the ingestion of nicotinic acid or the amide. It must be necessary for the

muscles also, and probably for the liver and some other organs, since it is an essential part of cozymase and of the co-enzyme of these tissues and organs."

Spies and his co-workers report the use of the Cenco-photolometer in determining the twenty-four hour output of nicotinic acid and have established 20 to 50 mg. as the normal for people having a well-balanced diversified diet.

Determinations of porphyrin have been made because it has been found in the majority of cases of pellagra studied by Sebrell and in many alcoholics by Jolliffe, and whenever present suggests the need for nicotinic acid.

In fact, I began using nicotinic acid empirically before a laboratory test was available, and have continued using it for an indefinite time whether the test is positive or negative because of the variability of the presence of porphyrin and because of increasing amount of eighth nerve degeneration observed in the rats and chicks deficient in the different fractions of the B complex, especially Jukes' chicks.

PORPHYRIN

Mason, Courville and Ziskind[26] state that porphyrin is found in the urine of photosensitive individuals (people who sunburn easily), chronic drug addicts (sulphonal, vironal), also in cases of lysol, chloroform, zinc and lead poisoning. Increased quantities of porphyrin are found in many febrile conditions and in certain types of disease of the liver, etc. It is present in Landry's paralysis, some cases of multiple sclerosis, and in almost 100 per cent of pellagrin cases, and a common finding in subclinical pellagra as well as eighth nerve deafness and conduction deafness.

Mason and his co-workers, describing the autopsy studies of their cases of Landry's disease, say: "The changes in the nervous system constitute the outstanding pathological lesion" and "extensive patchy degeneration of the myelin sheaths and axis cylinders, most marked in the nerves of the lower extremities."

Shattuck[27] views Landry's syndrome in the light of a nutritional disease and similar to beri-beri.

Porphyrin may not be present in the urine continuously, but when it does occur, it should not be disregarded as of no importance, even though the patient appears in perfect health.

LABORATORY METHODS

Thiamin Chloride: The method used in the determination of the twenty-four hour urinary thiamin output is a compilation worked out by R. K. Main, Ph.D., of the Harriman Research Laboratories, Southern Pacific General Hospital, and the analyses herewith reported were made by Doctor Main. The method used was a modification of the thiochrome method of Jansen and collaborators[28] using ferricyanide as the oxidant.[29] After oxidation of the thiamin to thiochrome in alkaline solution it is extracted with iso-butyl alcohol. The method as applied to urine by Westenbrink and Goudsmit[30] was used in a few determinations at the beginning of the series. The method found most satisfactory and used in all determinations of thiamin as reported in this paper, with the exception of the few mentioned above, was essentially that of Ritsert.[31] Two 5 cubic centimeter aliquots of the twenty-four hour urine specimen were made alkaline with 10 cubic centimeter portions of 15 per cent NaOH. One, the "unknown," was treated with 5 cubic centimeters of 1.0 per cent potassium ferricyanide solution, while the other, the "control," was diluted with an equivalent amount of distilled water. Both were shaken for two minutes. Each was extracted by continuous shaking for three minutes with 10 cubic centimeter portions of iso-butyl alcohol. After sufficient standing, each alcohol layer was pipetted off and filtered. The fluorescence of two cubic centimeter portions of the filtered alcohol layers, when illuminated at right angles to the line of vision by a beam of ultraviolet light, were compared. The control served to estimate the residual fluorescence due to preformed fluorescent substances in the urine. Portions, measured from a semi-micro burette, of a standard solution of thiochrome in iso-butyl alcohol, were added to the control until the fluorescence of the control and the unknown exactly matched. The amount of this standard solution added is a measure of the amount of thiamin originally in the urine. This standard solution was prepared from a standard aqueous solution of thiamin by an exactly similar manner to that used on the urine. The twenty-four hour urine specimens were kept acid by the presence of five cubic centimeters of glacial acetic acid placed in the collecting bottles. They were kept on ice until the determinations were made.

Porphyrin: The following method used in the determination of porphyrin in urine has been prepared by Jesse L. Carr of the Pathology Department, University of California Medical School, and the porphyrin analyses herewith reported were carried on in

Carr's private laboratory (Fitzhugh Bldg., San Francisco). To 25 cubic centimeters of urine add 0.5 cubic centimeters of acetic acid and 25 cubic centimeters of ether. Extract for twenty-four hours in a separatory funnel. To the ether fraction, add 25 cubic centimeters of 25 per cent hydrochloric acid which extracts the porphyrin from the ether fraction. This second extraction contains other chloride materials as well as porphyrin which can be removed with two to three washings of distilled water. The fluid is then placed in front of an ultra-violet light source filtered with a Woods filter. A pink fluorescence in the hydrochloric acid fraction is indicative of porphyrin, which is then identified in the spectroscope, the bands appearing about 450 and 500. In the urine and in the ether partition a blue fluorescence will be noted, which is due to vitamin B and other substances as yet unidentified.[32]

REPORT OF CASES

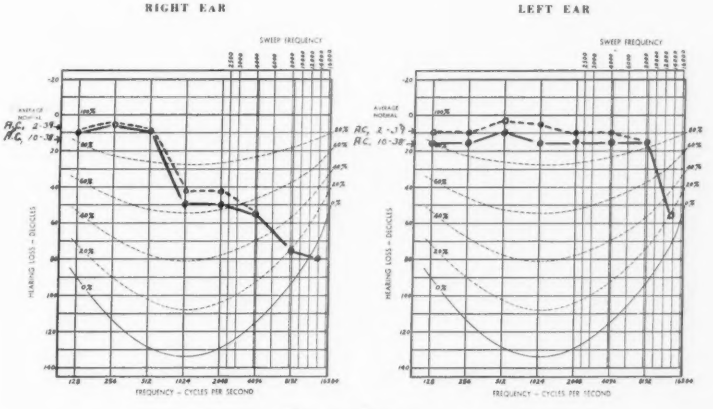


Fig. 2—Case 1.

CASE 1.—Patricia E., age 9. October 13, 1938.

Complaint: Deafness, right ear.

History: Deafness. Deafness followed an abscess in right ear at age of six years which discharged for one week. Both grandparents are deaf mutes; her grandmother had smallpox and diphtheria at age of one year; her grandfather fell from a high chair at age of one and one-half years and fractured skull.

Teeth: Good; has separated central incisors.

Allergy: No history of hay fever or asthma.

Diet: Mother's diet during the first six months of pregnancy was definitely out of line, not at all like Bogert's and Porter's diet for pregnant mothres, i. e., "The pregnant mother should have a diet rich in carbohydrates with only moderate amounts of protein and fat, but very rich in vitamins and mineral elements." They advocate one quart of milk, two eggs, meat sparingly, cheese, nuts and legumes, cereal foods such as wheat hearts, liberal use of fruits, including oranges and grapefruit, vegetables and leafy salads. The mother's diet was more correct during the last three months of her pregnancy. After birth, the child's diet was satisfactory until she was three years old. From the third year to the ninth, the diet has been very poor.

Diseases: Whooping cough at age of 5: mumps at 8; measles at 9.

Operations: Tonsils and adenoids removed at age of 4.

Examination—Physical: General health is good but is subject to frequent colds with bronchitis, and also subject to constipation. Patient has quite a lot of hair on her back.

Height: Height is 2½ inches less than span.

Ears: Left, normal drum; right, slight retraction of drum.

Nose: Slight post deviation of septum to the right.

Throat: Small amount of adenoid above both eustachian tubes; tonsils out.

Laboratory Data:

B.M.R.: Plus 10 per cent.

Urine: Porphyrin, negative.

Blood calcium: 12.1 mg. per 100 cc.

Blood phosphorus: 321.

Vitamin C: 0.8 mg. per cent.

X-rays: Wrists for bone age—normal. Skull—calcium content within normal limits. Sella—normal.

Treatment: October 15, 1938. Patient's diet is to be corrected—plenty of milk, wheat germ, citrus fruit, vegetables and proteins. In addition, she is to take the rice bran vitamin B complex, 1 teaspoonful t.i.d. (to February 24, 1939). Also carotene, 10,000 units daily, and C vitamic acid tablets, 50 mg. daily (to February 24, 1939).

February 24, 1939: Continue vitamin B complex; also carotene. Stop C vitamic acid tablets.

Comments: This case is reported as an illustration of Crowe's comment, i. e., that a drop in the high part of the tone scale is observed early in life. On February 24, 1939, patient's weight was 82 pounds; height, 4 feet 6 inches; span equals height; legs, 26½ inches; torso, 27½ inches. It is probable that the growth factors in her diet are responsible for the body changes.

Comparing the mother's diet during her first six months of pregnancy with Bogert's and Porter's diet for pregnant mothers, it is obvious that her diet was lacking in vitamins B and C. This lack during pregnancy and the child's unsatisfactory diet between the age of three and nine, plus her obvious pituitary disturbance, offers some evidence that such deficiencies may play a definite part

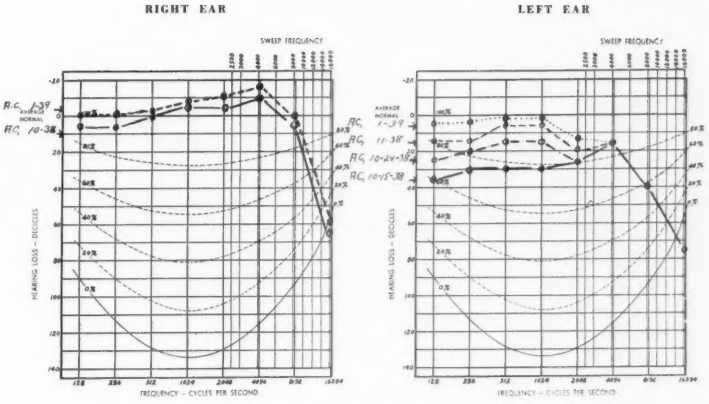


Fig. 3—Case 2.

in her loss of hearing in the right ear as well as the loss of high tones in the left ear.

The cold catching tendency has lessensed in a very striking way. The child voluntarily said, "My colds, instead of lasting four or five weeks, last no longer than one week." She also announced that she could hear better. The audiogram shows a slight improvement. There also appears to be a definite decrease in the size of adenoid remnants above both eustachian tubes.

Case 2.—Miss Jean D., age 18. October 15, 1938.

Complaints: Deafness, left ear; tinnitus.

History: Deafness. First noticed hearing impairment of left ear 18 months ago, at which time she also noticed a tinnitus that has been continuous, and varies from a low noise (sea shell) to an occasional high-pitched sound. This noise in the left ear followed the extraction of a tooth, and about that time she had spells of nausea and dizziness. Within the past four weeks, she has noticed a tinnitus of the right ear accompanied by a recurrence of the nervousness, nausea and dizziness. Grandmother was deaf.

Allergy: No history of hay fever or asthma.

Diet: Diet has been very poor. Low in vitamins B and C. No citrus fruit until the last year or two.

Catamenia: Began at age of 12 years (1932). At that time she noticed difficulty in keeping herself warm. During 1934 and 1935, she began to have nervous reactions and severe nausea, dizziness and cramps throughout her period lasting six days. In 1935 and 1936 she took thryoid for the menstrual disturbance and a bad complexion. In 1936 and 1937, her spells of nervousness and

painful menstruation gradually lessened and seemed normal. The last menstrual flow was profuse and again accompanied by nausea and dizziness.

Operations: Tonsils and adenoids removed last year (1937).

Diseases: Chickenpox during childhood.

Examination—Physical: Her internist consultant reports the following: Nothing of significance except a chronically dry skin, perpetual cold hands and feet, low morning pulse (40-50), and subnormal temperature.

B.P.: 116/72.

Weight: 135 pounds.

Height: 5 feet 5 inches.

Ears: Slight retraction of the drums. Rinné positive, both ears.

Nose: Normal.

Throat: Tonsils out.

Tubes: Open.

Hearing: Deafness appears to be mixed but mostly characteristic of nerve deafness involving principally the left ear, although there is a marked drop of high tones in the right ear (beyond 8192 cycles).

Laboratory Data:

B.M.R.: Normal.

Urine: Vitamin B_1—18.6 I.U./24 hours; porphyrin—negative.

Vitamin C: 0.74 mg. per cent (0.8 to 1.4 mg. per cent is normal).

Treatment: The otologist who referred this patient to me gave her thyroid, four grains daily, with little or no results; thiamin chloride, 4000 I.U. daily, for three weeks with no obvious improvement.

I gave her 150 mg. of C vitamic acid daily for seven days; then 50 mg. daily for three weeks, at which time a second audiogram was made. Following this (November 25, 1938), her physician gave her parenterally thiamin chloride, 67 mg., and nicotinic acid, 15 mg. weekly. Along with this there is to be an increase of protein intake in her diet as well as increase in the foods containing B_1 and C; then recheck blood and urine to determine if these substances are being recovered from her food intake.

Comments: This patient presents many interesting things: Her very low pulse, poor circulation, subnormal temperature, low B_1 output in spite of fairly large doses of thiamin chloride and low blood vitamin C.

Her original audiogram was identical to two done on my audiometer. The improvement in the hearing curve following a short course of C vitamic acid is rather striking, especially as no improvement followed the use of B_1 by mouth. Another striking thing is a further improvement shown in the audiogram of January 20, 1939, following the parenteral use of thiamin chloride and nicotinic acid.

This case is an example of several vitamin deficiencies occurring in one individual, i. e., vitamin C, thiamin chloride (B_1), and nicotinic acid (anti-pellagra factor).

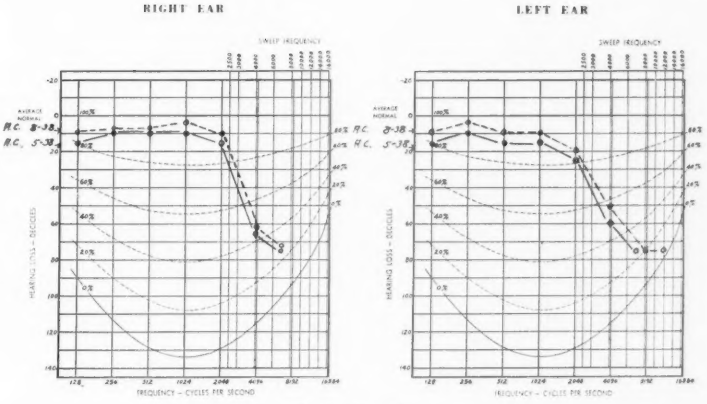


Fig. 4—Case 3.

CASE 3.—Mr. H. P. F., age 42. May 9, 1938.

Complaints: Deafness—both ears.

History: Deafness: Patient first noticed deafness several years ago. His mother has a nerve deafness.

Allergy: No history of hay fever or asthma.

Diet: Nothing of note except a definite vitamin B deficiency and too large an intake of carbohydrates.

Diseases: Measles, mumps, and whooping cough in childhood; influenza in 1918.

Examination: Physical: General health good. Smokes a lot and is very generous user of alcohol.

Weight: 145 pounds.

Height: 5 feet 10¾ inches.

Ears: Nothing unusual.

Nose: Normal.

Throat: Tonsils infected.

Tubes: Open.

Laboratory Data:

B.M.R.: May 10, 1938—minus 18 per cent; August 9, 1938—minus 13 per cent.

Urine: Vitamin B_1—11.1 I.U./24 hours; porphyrin—plus one.

Blood Count: Hgb. 98 per cent; R. B. C. 5,280,000; W. B. C. 9000.

Spectrographic Study of Ashed Blood (August 9, 1938): Fe, B, Si, P, Mg, A1, Ca, Ag, Na, K, Cu, Ba, Sa, As.

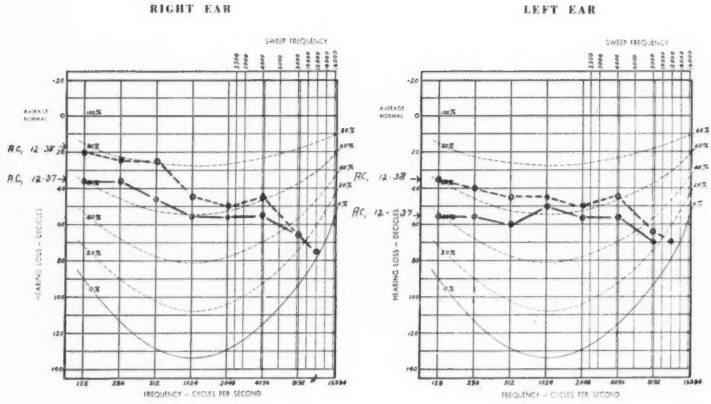


Fig. 5—Case 4.

Treatment: May 12, 1938: Armour's Thyroid, two grains daily (to August 3, 1938).

Comments: On August 3, 1938, there was a five decibel improvement in both ears. This patient, who lived in Honolulu, was advised to have his physician give him injections of thiamin chloride, 3000 I.U. per cc., one cc. six days a week for four weeks, then reduce the dose to 1000 I.U. and add nicotinic acid, one grain, t.i.d., by mouth, for three months along with the thyroid. Also to make a careful search for the source of the arsenic shown in the blood. This procedure apparently was disapproved of by the patient's physician for reasons unknown, since no further communications have been received from either the patient or physician.

Case 4.—Mr. A. J. H., age 59. December 2, 1937.

Complaints: Deafness—both ears.

History: Deafness: Had noticed deafness for several years. Is confused when several people are talking, especially when in conference. (Is employed by the Southern Pacific Company, and travels on railroads approximately 40,000 miles a year—for 20 years.) Tinnitus is like falling water; was first noticed in 1932. Had earache as a child. No history of colds.

Allergy: No history of hay fever or asthma.

Diet: Diet was very poor in childhood and adolescence. Definitely low in vitamins B and C. Diet is better at present.

Diseases: Had mumps and scarlet fever in childhood.

Examination: Physical: Is in excellent health. Recent physical examination shows nothing. Has occasional short colds. No tobacco; very little alcohol.

Weight: 160 pounds.

Height: 5 feet 8½ inches.

Ears: Nothing unusual.

Nose: Slight deviation of septum to right.

Throat: Tonsils are small.

Tubes: Open.

Laboratory Data:

B.M.R.: Minus 14 per cent.

Urine: Negative.

Wassermann: Negative.

Treatment: March 3, 1938: 25 injections nicotinamide, 30 mg. per cc. (to April 26, 1938).

April 26, 1938: Stop injections. Given nicotinic acid, two grains daily by mouth (to November 1, 1938).

November 1, 1938: Urine tested for vitamin B_1 output showed 8.1 I.U. per 24 hours.

November 7, 1938: Thiamin chloride injections, 3000 I.U. per cc., one cc. six days a week. Continue nicotinic acid, two grains night and morning (to date, February 10, 1939; is to be continued for several more months).

Comments: No improvement in audiogram was observed until April 26, 1938, at which time both ears showed a gain of five decibels throughout the scale. On July 12th, there was improvement of five decibels in several notes of both ears. On October 17th there was no further change from the chart of July 12th in either ear. Following the addition of thiamin chloride on November 7th, an audiogram on December 22nd, showed a further step-up of five or ten decibels in all notes of the right ear.

This case shows the dual nature of the deficiency and illustrates the advisability of trying the fractions singly, and carrying them on together in varying doses the balance of the natural life. It also shows that a process beginning at least at the age of forty in a man subject to the constant noise of a railway train can be definitely improved. And while a ten to twenty decibel gain may appear small, to a railroad trouble-shooter, the go-between in labor union conference disputes, it meant the difference between continuance of a valuable job or retirement.

CASE 5.—Mr. E. V. R., age 60. May 24, 1938.

Complaints: Deafness—Both ears; tinnitus.

History: Deafness: Noticed deafness in left ear at age of 6 or 7 years, following a fall from a horse. Had abscess in left ear at age of 9. Noticed deafness in right ear at age of 26 or 27 years. Noticed no further change in hearing until one year ago, when hearing seemed to decline and tinnitus increased. Has con-

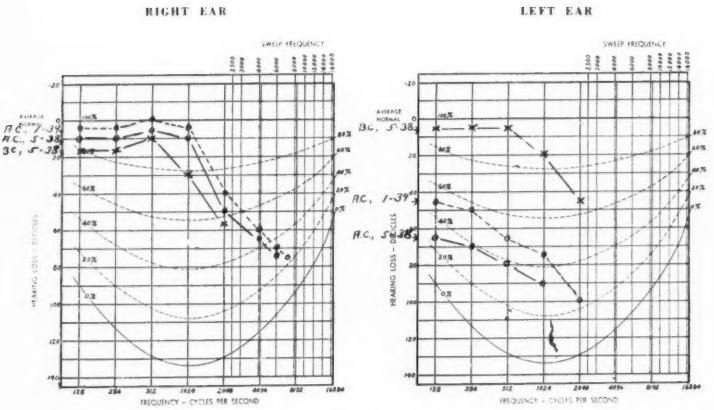


Fig. 6—Case 5.

stant tinnitus in right ear, ringing in character. Patient's mother was deaf after age of 50 years.

Allergy: No history of hay fever or asthma.

Diet: Prior to ten years ago, his diet was always very poor, with very little vitamin B and C intake in winter time. His diet the last five years has been better.

Operations: Appendix and hernia.

Diseases: Had all children's diseases; severe case of influenza ten years ago.

Examination: Physical: General health good except for constipation and sensation of fatigue on waking and during the day.

B.P.: 125/65.

Weight: 144 pounds.

Height: 6 feet.

Ears: Slight retraction and light spot not quite normal in right; marked retraction in left.

Nose: Septum has slight deviation.

Throat: Tonsils present; no evident infection.

Tubes: Open.

Laboratory Data:

B.M.R.: Minus 7 per cent.

Blood Count: Hgb. 99 per cent; R. B. C. 4,940,000; W. B. C. 7,900.

Treatment: May 24, 1938: Nicotinic acid, one grain, t.i.d. (to September 15, 1938).

September 15, 1938: Sodium nicotinate, two grains, night and morning (to October 24, 1938).

October 24, 1938: Nicotinic acid, two grains, night and morning; and thiamin chloride injections six days a week, 1000 I.U. per cc. (to January 25, 1939).

January 25, 1939: Discontinue thiamin chloride and nicotinic acid for one month, at the end of which time another audiogram is to be made.

Comments: This patient apparently had in the left ear an obstructive deafness, probably adhesive in character, with so-called secondary nerve changes; and in the right ear a definite nerve involvement.

On October 24, 1938, before thiamin chloride was added, three notes of the right ear had improved five decibels; and four notes of the left ear had improved 20 decibels. On January 25, 1939, after the patient had been taking thiamin chloride for three months, three more notes of the right ear had improved five decibels; and note 2048 was now audible by the left ear. Here it also seems that the increase in hearing in a man of 60 years of age is due to nicotinic acid plus thiamin, and illustrates the dual nature of the deficiency.

CASE 6.—Mr. J. B., age 65. April 30, 1938.

Complaints: Deafness—both ears.

History: Deafness: First noticed deafness in right ear at age of forty. In 1928, he had an attack of vertigo with staggering when he walked and was sick at his stomach. He was confused when several people were talking. He hears fairly well over the telephone. Patient has no tinnitus. One sister is deaf; hearing of both parents was good.

Allergy: Has frequent colds, possibly allergic.

Diet: Definitely low in vitamins A, B and C. Has little milk, few eggs, infrequent salads, occasional citrus fruits, but plenty of vegetables and fruits in season.

Operations: Tonsils removed several years ago and also an intranasal operation on the right antrum. (Relief from the antrum lasted one year with frequent recurrent infections, probably allergic in character.)

Examination: Physical: His physician reports a low-grade prostatitis, a variable blood pressure (150 to 200), the usual amount of arteriosclerosis, some slight evidence of cardiac damage, a somewhat unstable vasomotor system, and constipation for years.

Weight: 183 pounds.

Height: 6 feet 1 inch.

Ears: Normal.

Nose: Normal.

Throat: Normal; postnasal discharge, occasionally yellow in color; tonsils out.

Tubes: Open.

Hearing Tests: Whisper, right, 3 inches; left, 4 inches. Conversation, right, 18 feet; left, 24 feet.

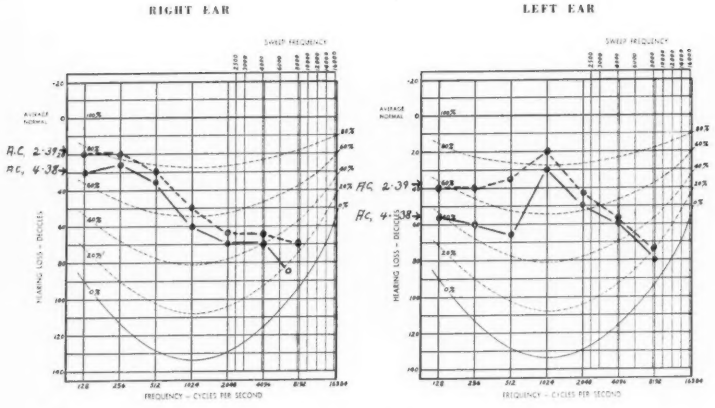


Fig. 7—Case 6.

Laboratory Data:

B.M.R.: Minus 11.5 per cent.

Blood Count: Hgb. 91 per cent; R. B. C. 5,310,000; W. B. C. 5,800.

Treatment: May 1, 1938: Nicotinic acid, one grain, t.i.d. (to February 6, 1939).

August 3, 1938: One cc. injection of thiamin chloride daily, 3000 I.U. per cc. (to November 1, 1938). Continue nicotinic acid capsules.

November 1, 1938: Reduce dose of thiamin chloride; continue nicotinic acid.

February 6, 1939: Given capsules containing 2000 I.U. of B_1 and 2 grains of nicotinic acid daily.

Comments: On November 1, 1938, having had thiamin chloride parenterally for ten weeks, this patient's constipation was almost entirely relieved and there was a definite improvement in hearing and general health. His urinary output of B_1 had increased to 69.5 I.U./24 hours (normal: 20-35 I.U./24 hours). This case illustrates not only the dual character of the deficiency, i. e., B_1 and nicotinic acid, but also the length of time required to saturate the body cells with thiamin chloride (ten weeks) before the output was doubled.

On February 6, 1939, following a reduction in the dose of thiamin chloride over a period of two months, the urinary output of B_1 was found to be 16.8 I.U./24 hours. This case well illustrates the fact that people of advancing years may need to take either these two substances together or the total B complex off and on the balance of the natural life.

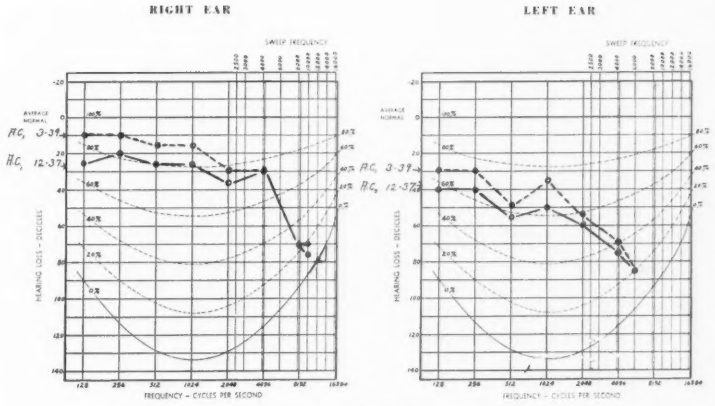


Fig. 8—Case 7.

Case 7.—Mr. W. S. B., age 73. December 2, 1937.

Complaints: Deafness of left ear; unsteadiness on his feet on arising in the morning, some mental confusion, and some loss of his sense of direction.

History: Deafness: Has difficulty in hearing conversation, especially when in conference or at a theatre. Difficulty in hearing was first noticed several years ago.

Allergy: No allergic history.

Diet: Dietary habits have always been poor.

Operations: Had operation on left antrum for a chronic infection.

Examination: Physical: Has been under the observation of his general physician since 1921 for an hypertrophic arthritis and a paroxysmal tachycardia and spells of dizziness. Arteries somewhat thickened. Some nutritional anemia. Is a moderate consumer of alcohol. Recently complained of neuritis in left shoulder (March 10, 1934), which disappeared after the extraction of two teeth.

B.P.: 145/75.

Weight: 135 pounds.

Height: 5 feet 9 inches.

Ears: Normal.

Nose: Normal.

Throat: Tonsils out.

Tubes: Open.

Laboratory Data:

Urine: Normal.

Blood Count: Hgb. 75 per cent; R. B. C. 3,480,000; W. B. C. 10,350.

Treatment: December 2, 1937: Seven injections of Lederle's B_2 complex (to December 29, 1937).

December 29, 1937: Nicotinic acid, one grain daily (to June 13, 1938).

March 1, 1938: Nine injections Riboflavin (to March 29, 1938). Continue nicotinic acid.

May 5, 1938: Ten injections thiamin chloride, 3000 I.U. per cc. (to June 13, 1938). Continue nicotinic acid.

June 13, 1938: Nicotinic acid capsules increased to one grain, t.i.d.

Comments: This patient has been a good "human guinea pig." He was willing to have tried all the available fractions of the B complex. His improvement followed nicotinic acid and B_1. This illustrates the observation that as the years advance, more nicotinic acid and thiamin chloride are necessary than found in the B complex rice bran or elsewhere unless given in very large doses.

While the hearing gain in both ears does not exceed 15 decibels, the improvement is positive. Twelve audiograms have been made since the first one. The improvement in his ataxic symptoms, confusion, etc., is very definite and by itself seems worthwhile. This improvement appeared to be due more to nicotinic acid and thiamin chloride. Similar improvement in another case, aged 75, showing mental confusion, etc., is equally striking.

B_1 was discontinued on June 13, 1938, and the nicotinic acid continued. On January 6, 1939, a recheck of the urine for the B_1 output showed 7.5 I.U. for the twenty-four hours, hence the use of B_1 will be renewed to be continued indefinitely, first parenterally for several weeks, then by mouth. He may need these materials more or less the balance of his life.

COMMENTS

Referring to case one, let me quote from Minot's[333] recent paper, as follows: "It is well to remember that since the nutritional states of the mothers affect the well-being of infants, the health of the whole population depends to a greater or less extent on maternal nutrition. Likewise, let us recall that at no period of life is optimal nutrition more important than in infancy and childhood, because nutritional defects so readily produced then may be at the root of disorder arising at any time later in life." Elsewhere in the same article he says: "The subtle effects produced by long standing slight nutritional defects need evaluation, but in practice they should be prevented if possible. In considering the far-reaching influences of faulty nutrition, it should be recalled that there is evidence that it may affect adversely the span of life and that the undesirable effect of a faulty diet in the zone of partial deficiency may become detectable only after years or generations, and that a deficient diet may impair the vigor and resistance of an individual for some period after he partakes of an adequate diet." These state-

ments of Minot's offer a satisfactory explanation of many of the symptoms complained of by many patients in the higher age brackets, notably case number seven.

Explanation is needed for the lowered basal metabolism rates seen in cases that are not frank hypothyroids. Certain laboratory studies (method of H. Jones[34]) are in progress at the Harriman Research Laboratories that seem to indicate that many cases are related to nutritional factors, principally to protein deficiency.

This study and the data presented herewith are intended to point the way to other factors involved in degeneration of the auditory apparatus.

Further study is necessary concerning the relationship of vitamin C and also what possible influence certain substances, possibly toxic in character, may have on the nerve as observed at the Harriman Research Laboratories of the Southern Pacific General Hospital by Arschambault in the spectroscopic study of ashed blood in a series of cases. In addition to calcium, phosphorus, potassium, magnesium and sodium, he found iron, boron, silicon, aluminum, silver, copper, barium, strontium, arsenic, titanium, cobalt, chromium, tin, antimony, zinc and lead.

It is important to know their source, how they gain access to the blood stream and whether damaging to nerve tissue. Such information of necessity will require a lot of animal experimentation and subsequent histological study.

A long period of time may be required before a full answer is at hand, and yet the evidence offered is in part at least an answer to the statement by the Harvey Lecturer that the cause of eighth nerve atrophy is unknown.

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REFERENCES

1. Crowe, S. J.: Underlying Causes of Deafness. Harvey Lectures, 1931-32.

2. Crowe, S. J.: Diagnosis and Differential Diagnosis of Deafness. Arch. Otol., 28:663-675 (Nov.), 1938.

3. Gray, A.: The Otosclerotic Problem. J. of Laryng. & Otol., 49:10 (Oct.), 1934.

4. Shattuck, G. C.: Nutritional Deficiency and the Nervous System. J. A. M. A., 111:1729-1734 (Nov. 3), 1938.

5. Selfridge, Grant: Can Certain Diseases of the Ear, Nose and Throat, Especially Degeneration of the Eighth Nerve, Be Classified as Deficiency Diseases? Laryngoscope (Feb.), 1936.

6. Selfridge, Grant: Eighth Nerve High Tone Deafness From a Nutritional Standpoint: A Preliminary Report. ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY, 46:1-93 (March), 1937.

7. Selfridge, Grant: Chronic Progressive Deafness From a Nutritional Standpoint: A Preliminary Report. ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY, 46:4-875 (Dec.), 1937.

8. Selfridge, Grant: Nicotinic Acid and the Eighth Nerve: A Preliminary Report. ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY (in press).

9. Covell, W. P., and Noble, L.: The Significance of Myeline Sheath Degeneration for the Cochlear Nerve. ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY, 46:895-911 (Dec.), 1937.

10. Williams, R., and Spies, T.: Vitamin B_1. First edition. Macmillan Co.

11. French, C.: Vestibular Function in Experimental Beri-beri. Proc. Soc. Exper. Biol. & Med., 30:1158-1160, 1933.

12. Shattuck, G. C.: Landry's Paralysis in Relation to Vitamin B Deficiency. International Clinics, 3:48, 1938. J. B. Lippencott Company.

13. Lewy, F. H.: The Pathology of Nerve Deafness. Trans. Am. Otol. Soc., p. 123, 1937.

14. Jolliffe, R., and Goodhart, N.: Effects of B_1 Therapy on the Polyneuritis of Alcoholic Addicts. J. A. M. A., 110:6 (Feb. 5), 1938.

15. Mellanby, E.: Experimental Production of Deafness in Young Animals by Diet. J. of Physiol., 94:380-398 (Dec.), 1938.

16. Ono, T.: Experimental Histopathological Studies of the Ears in A, B and C Avitaminosis. Trans. Japanese Path. Soc., 18:172 (Jan.), 1928.

17. Covell, W. P.: Personal Communication (unpublished).

18. Eddy, W., and Dalldorf, G.: The Avitaminoses. Williams & Wilkins Co., 1937.

19. Greenberg, D. M., and Tufts, E. M.: The Nature of Magnesium Tetany. Am. J. of Physiol., 121:2 (Feb.), 1938.

20. Greenberg, D. M.: Experimental Studies on Calcium. Personal Communication (unpublished).

21. Williams, R., and Spies, T.: Vitamin B_1. Macmillan Company, First Edition, page 731.

22. Cowgill, G. R.: Human Requirements for Vitamin B_1. J. A. M. A., 111:11 (Sept. 10), 1938.

23. Weiss, S.: See Jolliffe's and Goodhart's Article (ref. 14).

24. Steinberg, C.: Untoward Effects Resulting From the Use of Large Doses of Vitamin B_1. Am. J. Digest Dis., 5:680 (Dec.), 1938.

25. Vilter, S.; Spies, T., and Mathews, A.: A Method for the Determination of Nicotinic Acid, Nicotinamide and Possibly Other Pyridine-like Substances in the Human Urine. J. Biol. Chem., 125:1 (Sept.), 1938.

26. Mason, W.; Courville, C., and Ziskind, E.: Porphyrins in Human Disease. Medicine, 12:413 (Dec.), 1933.

27. Shattuck, G. C.: International Clinics. J. B. Lippencott Co., 3:48:26, 1938.

28. Jansen, B. C. P.: Rec. Trav. Chim., 55:1046 (1936).

29. Barger, G.: Todd, A. R., and Bergel, R.: Ber., 68:2257 (1935).

30. Westenbrink, H. G. K., and Goudsmit, J.: Rec. Trav. Chim., 56:803 (1937).

31. Ritsert, K.: Deutsch. med Wochschr., 64:481 (1938).

32. Fischer, H.: Neuere Methoden der Isolierung und Nachweis von Porphyrinen. Abderhaldens Handbuch der biologischen Arbeitsmethoden, Abt. I. Teil II, Lief. 211, 1927.

—u. M. Dezelic: Uber die Einwirkung von Ozon auf Porphyrine. Hoppe-Seylers Z. 222:270 (1933).

33. Minot, G.: Nutritional Deficiency. Annals Int. Med., 12:4 (Oct.), 1938.

34. Jones, H.: The Cause of Goiter. Monograph Publishers, Chicago.

LIII

A METHOD OF CLOSING ANTRO-ALVEOLAR FISTULAE*

REA E. ASHLEY, M.D.

SAN FRANCISCO

An antro-alveolar fistula may be defined as an opening persisting between the maxillary sinus and the mouth through the alveolar process. The majority of such fistulæ occur following tooth extraction and are cared for by the dental surgeon. Ordinarily they heal rapidly, but when complicated by infection in the antrum, by dental cysts, by malignancies or by great loss of tissue from the floor of the antrum, the technique of closure presents a real problem. An operation which has been successful in closing such fistulæ will be described later in this paper.

HISTORICAL NOTE

No historical data appear in the literature regarding this not uncommon condition.

Nathaniel Highmore,[1] in 1651, was the first to report a case of antro-alveolar fistula. In his work on human anatomy, he gives the case report in the following words: "The lower surface of this cavity *(antrum genae)* is very thin and frequently upon drawing a tooth is taken along with it, whereby the cavity is opened into the mouth. A gentlewoman who had the *dens caninus* drawn on account of inveterate defluxion of sharp humor, on thrusting a silver bodkin into the alveolus was exceedingly frightened to find it pass, as it did, almost to her eyes. Upon further trial with a small feather stripped of its plume, she was so terrified as to consult a doctor and others, imagining nothing less than it had gone to her brain. But they, considering the circumstances, found that the feather had doubled only into this cavity."

Some years later, in 1707, Cowper,[2] an anatomist, asserted that Highmore's case had suggested to him the operation of alveolar drain-

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age of the sinus which he described in Drake's Anatomy. Many textbooks still advocate Cowper's operation as a possible method of drainage, but the operation lacks scientific basis, and the results have been so uniformly unsatisfactory that most rhinologists agree with Voorhees,[2] who described it as "the worst operation in surgery, converting a simple condition into a chronic suppuration."

The later literature, which appeared from Cowper's time to early in this century, dealt not with methods of closing fistulæ, but with methods of establishing and maintaining a patent fistula from an infected antrum into the mouth.

Mention of the condition was made by Heath[4] in his work "Injuries and Diseases of the Jaw," published in 1890, in which he remarks: "In spite of all treatment, some of these fistulæ refuse to heal." In 1920, Welty[5] described a plastic operation as a means of closing such fistulæ. Since that time articles have appeared dealing with various phases of the subject by Guthrie,[6] Brown,[7] Lyons,[8] Price,[9] Maybaum[10] and others.

ANATOMICAL RELATIONSHIP

Before discussing the etiology of this condition, it is of interest to review briefly the anatomical relationship between the floor of the antrum and the teeth, since this relationship has a direct bearing on the development of fistulæ.

Morris[11] described the floor of the antrum as follows: "The floor of the sinus is usually very uneven due to the prominences corresponding to the roots of the molar teeth. In most cases, the bone separating the teeth from the sinus is very thin and occasionally the roots project into it. The teeth which come into the closest relation with the sinus are the first and second molars, but sockets of any of the teeth lodged in the maxilla may, under diseased conditions, communicate with it."

Mustian[12] from an anatomical study of one hundred maxillary sinuses, drew the conclusion that, while there is considerable variation, the sinus floor usually extends from the second bicuspid to the infra-temporal wall of the maxilla. This explains the fact that fistulæ most frequently follow extraction of the first or second molar or the second bicuspid.

His observations also showed great variation in the relationship of the roots of the teeth to the antral floor. In his specimens the apical ends of the roots as compared to the floor of the sinus varied as much as twenty millimeters in their extremes. Certain of his

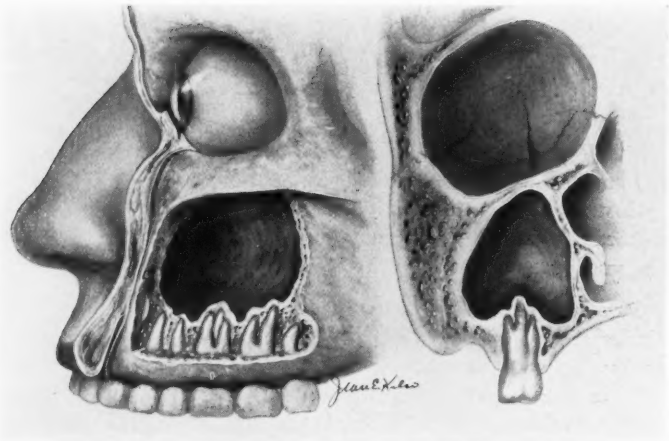

Fig. 1.

dissections showed as much as ten millimeters of process between the apices of the roots of the teeth and the floor of the sinus, while others showed the floor of the antrum descended to the bifurcation of the molars, leaving the roots of these teeth ten millimeters above the floor in the walls of the sinus.

The pneumatization of the maxilla is an important predisposing factor in the formation of alveolar fistulæ, that is, the more complete the pneumatization, the greater the number of teeth which extend to the floor of the antrum, and the thinner the process between the roots of the teeth and the antral cavity (Figs. 1 and 2).

ETIOLOGY

The etiological factors in the development of a fistula into the maxillar are dental necroses, failure of operative technic directed to the maxilla, pathology in the antrum, and, rarely, dental cysts, malignant tumors, trauma and foreign bodies.

The alveolar type of fistula is usually of dental origin. An area of infection and necrosis first arises at the root of an upper molar or premolar tooth, and then, by extension, the alveolar ridge and the floor of the antrum become involved. When the infected tooth is extracted, it is found that either the roots extend up into the antrum

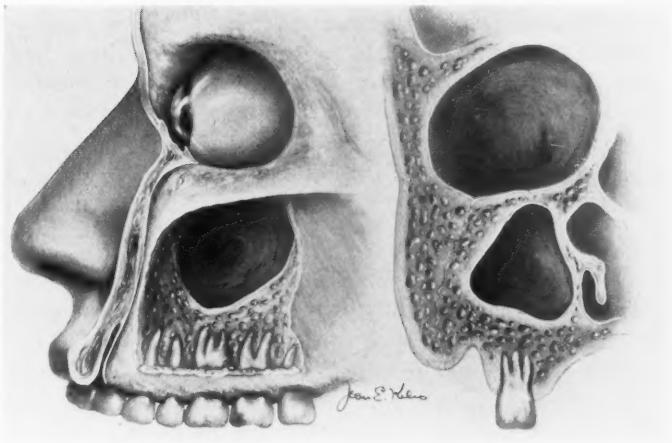

Fig. 2.

or that the necrotic process has become so extensive as to involve the floor of the sinus. In either case, a fistula results.

Failure of operative treatment is a frequent cause of fistula formation. The antrum is often accidentally exposed by the extraction of an adjacent tooth. The intervening bone over the bifurcation of the first and second molars is sometimes so paper-thin and fractures so easily that extraction causes the removal of the tooth and the alveolar process en masse. This accident is serious only if the overlying antrum is infected or if the lining membrane of the sinus is opened. Over-energetic curetting and carelessness in probing the alveolar socket is often the direct cause of this complication.

Once communication is established between the antrum and the normal flora of the mouth, infection of the antrum follows immediately. The problem then is no longer that of dealing with a fistula, but with a fistula complicated by an acute sinusitis. Any attempt to force fluids through the alveolar opening or otherwise treat the infected antrum through this opening serves only to spread the infection and is not without danger to the other sinuses and to the middle ear.

Antral infection is another frequent cause of antro-alveolar fistulæ. For some years there has been much controversy as to

whether infection in the maxillary sinus is the cause of involvement of the roots of the adjacent teeth or whether the reverse is true. It is probable that infection occurs in both directions. Regardless of how an antrum becomes infected, the extraction of an underlying tooth is often followed by a fistula from the antrum into the mouth.

Dental cysts are somewhat rare, but occasionally do cause antro-alveolar fistulæ. They are divided according to their own origin into periodontal, dentigerous, and multilocular cystomas.

The periodontal or root cyst has its origin in the periodonteum of the root of a diseased tooth. Once the cyst has developed, it can continue to grow independently of the tooth, and by pressure necrosis, completely destroy the upper jaw or rupture into the antrum, the nose or the cheek.

Dentigerous cysts are rare and are due to an error in the development of the follicle prior to the eruption of the teeth. They occur most often during second dentition and usually contain one or more teeth or rudiments of teeth.

Multilocular cysts are usually of malignant origin.

TYPES OF FISTULAE

Antro-alveolar fistulæ may be classified as follows:

1. Small fistulæ into the antrum which do not become infected. These usually heal readily and may require at most a suture to pull the soft parts together.

2. Small fistulæ which granulate rapidly but do not close spontaneously. These may be so small as to defy probing and yet large enough to allow a constant flow of foul-smelling discharge into the mouth. Osteomyelitis of the maxilla, cysts, root fragments and sequestrae or overlying antrum infections may be the cause. Unless an overlying antrum infection is present the treatment of the condition is best left to the oral surgeon.

3. Large fistulæ which may granulate in and become small, but which are complicated by antrum infection. These require close cooperation between the oral surgeon and the rhinologist. If infection in the antrum remains after the fistula has been closed, it may go undiagnosed for years, giving all the general symptoms of focal infection.

4. Large fistulæ which persist as large openings into the antrum with a great loss of tissue both from the alveolar ridge and the floor of the antrum. These can be closed only by a plastic operation.

SYMPTOMS

Patients suffering from antro-alveolar fistulæ complain of many disagreeable symptoms. If the opening is large, food and liquids are forced up into the antrum and nose during mastication and swallowing. In many cases a whistling or hissing noise is present during conversation. When infection is present in the antrum, there is a purulent discharge into the mouth, and the patient is conscious of a foul odor in the nose and a bad breath at all times. Tenderness over the involved antrum and headache are frequent symptoms.

DIAGNOSIS

The presence of antro-alveolar fistula can usually be established by inspection and palpation with a probe and confirmed by means of an x-ray examination. If the fistula is very small, however, the diagnosis is not necessarily so simple a matter. In such cases x-rays following the injection of the fistulous tract with some radiopaque substance is of great value in determining whether or not there is a direct connection between the mouth and the antrum. Not infrequently the patient will make the diagnosis himself by drawing secretion from the antrum into the mouth by sucking.

TREATMENT

The treatent of alveolar fistulæ depends chiefly upon the size of the fistula, its duration and upon the presence or absence of infection in the antrum. The usual procedure mentioned in the early literature was to insert a wooden plug into the hole temporarily until a silver or gold cannula could be made to fit the tract. This latter was worn indefinitely and was held in place by attachment to the adjacent teeth. The patient was taught to remove the plug at frequent intervals to allow the nasal secretions and pus from the antrum to discharge into the mouth.

Fortunately, when a fistula occurs following the extraction of a tooth, the antrum does not always become infected. Cogswell[13] says, "It is highly important to avoid rupture of the membrane (of the antrum), as its unruptured continuity prevents entrance of pathological material into the antrum." If a perforation does occur, however, he advocates immediate closure at the time of extraction.

Closure is accomplished by removing sufficient of the buccal and lingual plate to permit approximation of the buccal and lingual flaps for suturing without tension. Cogswell believes that this not only effectively closes the socket until regeneration takes place, but that much antral irritation and infection can thus be avoided.

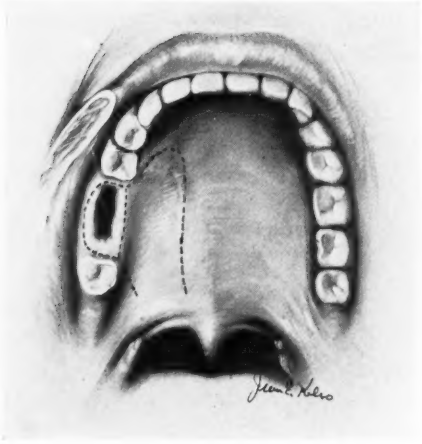

Fig. 3.

If pus is found draining from an alveolar pocket several days following extraction, it is probable that a fistula has resulted, and that the antrum has become infected. Diagnosis can be verified by x-rays and by gentle probing of the antral floor. If x-rays show only a fistula and no marked thickening of the membrane lining the antrum, a large window should be cut between the antrum and the nose through the medial wall under the inferior meatus. This opening should be kept patent by frequent shrinking of the inferior turbinate and by suction. Irrigation of the antrum through the tooth socket or through the inferior meatal window is counter-indicated.

If the fistula is large, and if the x-ray reveals the presence of marked thickening of the mucous membrane lining the antrum, complete antrum operation and plastic closure of the fistula should be performed as soon as the acute symptoms subside.

The sooner adequate drainage from the antrum into the nose is established and the sooner the opening into the mouth is closed, the shorter will be the convalescence and the less radical will be the treatment required.

Experience shows that a fistula will not close if pus is being formed in the antrum unless some easier path of drainage is established.

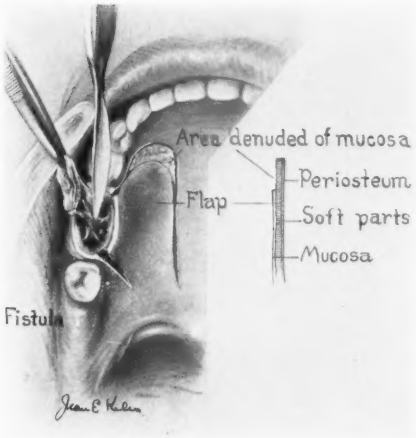

Fig. 4.

When a fistula fails to close following the usual simple methods, conservative treatment is of little avail. Packing over a long period stimulates the ingrowth of epithelium into the fistula and tends to make the opening permanent. Curetting or removing more bone only increases the size of the fistula. Drury[14] reports six of seven cases successfully treated by daily applications of liquor epispasticus, and Weih[15] reports six cases cured by repeated application of trichloracetic acid, but, while the use of escharotics may ultimately close the fistula, such treatments, even though continued over a long period of time, do not always effect a cure, and, furthermore, there is great danger of producing changes in the mucosa and chronic antral infection.

Failure of conservative treatment has led to the development of more radical plastic methods.

TREATMENT BY PLASTIC OPERATION

The choice of operation best adapted for plastic closure of any fistula is determined by the size and location of the fistula, since no one technic is applicable to all cases.

In small fistulæ Zange's[16] operation is very satisfactory. He makes a folding-door incision, undermines the mucosa on both sides

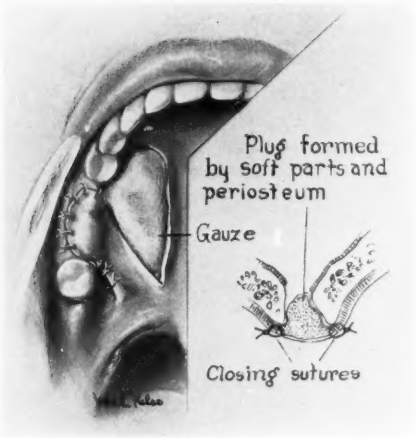

Fig. 5.

and levels the fistulous bone down to the floor of the maxillary sinus. The lining of the fistula is turned inward and the flaps sutured.

Axhausen's[17] method does not sacrifice the alveolar crest. He excises the fistulous tract and covers the defect with a musculomucosal flap from the cheek. Lautenschlager's[18] method makes use of a bone flap.

Larger fistulæ can be closed by utilizing a wide flap of mucoperiosteum from the palate by making a median incision, Welty's[15] operation, or by swinging a palatal flap into the opening as suggested by Dunning.[19] A similar method is described by Mead.

Following the same principles as those which I described for closure of postauricular fistulæ,[20] it has been possible to close several large antro-alveolar fistulæ in which other less radical methods had failed.

TECHNIC OF OPERATION

1. Infection in the antrum is controlled by the Caldwell-Luc operation.

2. The fistulous lining is removed, and any necrotic bone is curetted away.

3. The edges of the fistula are undermined to the bone and the alveolar ridge made smooth.

4. A flap is cut from the palate approximately one-third larger than necessary to close the fistula. The flap, including soft parts and periosteum, is attached by a wide pedicle toward the soft palate to insure adequate blood supply.

5. The flap is now swung into place and the distal end which has been denuded of its mucous membrane is wedged into the freshened fistulous tract, plugging it tightly

6. The mucous membrane edges of the flap are approximated to the mucous membrane edges of the fistula with black silk sutures (Figs. 3, 4 and 5).

This operation has the following advantages:

1. It is especially adapted to the closure of large fistulæ.

2. The plastic operation can be combined with the Caldwell-Luc in one operation.

3. The thick plug of tissue makes a firm closure and minimizes the tendency of the flap to break down. It also tightly closes the communication between the mouth and the antrum, thus encouraging healing by first intention.

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BIBLIOGRAPHY

1. Highmore, N.: Corporis Humanis Disquisitio Anatomica; published 1651, pp. 226-228.

2. Cowper, Wm.: Drake's Anthropologia Nova; published 1707, XI, pp. 534-537.

3. Voorhees, I. W.: Bucco-antral Fistula: Closure by Plastic Procedure. Laryngoscope, 37:431-432, 1927.

4. Heath, Christopher: Injuries and Diseases of the Jaw; published 1894 (4th Edition).

5. Welty, Cullen E.: Closure of Fistulous Openings Through the Alveolar Process Into the Antrum of Highmore. Trans. Section of Laryngol., A. M. A., published 1920, p. 198.

6. Guthrie, Douglas: Operative Closure of Oro-Maxillary Fistula. J. Laryngol. & Otol., 47:459, 1932.

7. Brown, Lewis E.: Relationship of the Teeth to Pathology in the Maxillary Sinus. Annals of Otology, Rhinology and Laryngology, 34:150 (Mar.), 1925.

8. Lyons, Horace: The Treatment of Alveolar Fistulæ Secondary to Extraction of Teeth. Trans. of Amer. Laryngol., Rhinol. & Otol. Soc., p. 81, 1925.

9. Price, M. J.: Progressive Medicine; published March, 1924, pp. 272-274.

10. Maybaum, J. L.: Two Cases of Empyema of the Maxillary Sinus of Dental Origin, Alveolar Fistula Radical Plastic Operation. ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY, 33:139 (Mar.), 1923.

11. Morris, Henry: Human Anatomy; published 1921, Blackstone.

12. Mustian, Wallace F.: The Floor of the Maxillary Sinus and Its Dental, Oral and Nasal Relations. J. Amer. Dental Assoc., pp. 2175-2187 (Dec.), 1923.

13. Cogswell, Wilton W.: Dental Oral Surgery; published by Dental Press, Inc., p. 309, 1932.

14. Drury, Dana W.: A Procedure for the Cure of Antral Fistulæ. Boston Med. & Surg. J., p. 1123 (Dec.), 1924.

15. Weih, E. P.: Closure of Alveolar Fistulæ by Scar Tissue Formation. ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY, 39:534 (June), 1930.

16. Zange, J.: Zum Plasticschen Verschlusse Von Kieferhohlenmundfisteln. Beitr. Z. Anat., Physiol., Path. u. Therap. d. Ohres., etc., 23:685-692, 1926.

17. Axhausen, G.: Der Plastische Verschluss der Antrum-Mundhohlen-Verteindungen. Deutsche Monatschr. F. Zamh., 28:193-204, 1930.

18. Lautenschlarger, A.: Kirschner's Operative Surgery by Ravdin. First Edition, Lippincott, p. 114.

19. Dunning, Henry Sage: Maxillary Sinusitis of Oral Origin. Laryngoscope, p. 766 (Oct.), 1925.

20. Mead, Sterling: Oral Surgery. C. V. Mosby Co., 1st Edition, p. 780, published 1934.

21. Ashley, Rea E.: Postauricular Fistula. ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY, 46:477 (June), 1937.

LIV

THE MANAGEMENT OF CICATRICIAL STENOSIS OF THE LARYNX*

JOHN H. FOSTER, M.D.

HOUSTON, TEXAS

So much has been written on this subject by the masters of laryngology that it would be presumptuous for me to think that I can improve materially upon the methods advocated by Jackson, von Schmiegelow, St. Clair Thomson, Negus, Figi, Arbuckle and others of greater knowledge and wider experience than I possess.

There are three reasons for the presentation of this subject:

1. To stress the fact that the great majority of cases are amenable to treatment.
2. To discuss the adaptability of the various methods advocated to the conditions encountered in private practice.
3. To offer an anatomical suggestion which seems to me to have an important bearing on the handling of a certain class of cases.

It has been my observation that many men doing laryngology are rather chary of attempting measures for the relief of these cases. Lack of experience, and perhaps difficulties encountered in attempted cases, breed timidity. A tendency to let well enough alone has strong authority, as evidenced by the following quotation from Thomson's book on Diseases of the Nose and Throat: "When it is recollected that patients wearing a tracheotomy tube may live fifty and seventy-five years, as described on page 885, in good health and vigor, and in the enjoyment of what voice they have, we should pause in many cases before embarking on treatment which may be tedious, trying and often unsatisfactory." Of course, those familiar with his work know that Sir St. Clair did splendid work for the relief of many of these cases.

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*Presented before the Sixty-first Annual Meeting of the American Laryngological Association, Rye, N. Y., May 24, 1939.

No attempt will be made to review the work done by various surgeons or the bibliography of the subject. My experience in dealing with cicatrical stenosis of the larynx is not particularly great, but it has extended over a great many years. I have tried the various methods, have encountered most of the difficulties and complications to be met with in handling these cases, and have formed some rather definite ideas and conclusions which are offered for whatever they may be worth. The conditions obtaining in private practice differ in some important respects from those associated with a public clinic. Many of the patients are from a distance. Most of them are of limited means and, whether or not, it is not convenient, or possible, for them to remain long under continuous observation. The economic aspect of the problem is doubly acute in the case of a working man who must be returned to his employment in the least possible time.

ETIOLOGY

Almost all cases can be classed as due to one of the following causes:

1. Corrosive chemicals are an infrequent cause, but they do occur. I shall refer to a case later.

2. Impacted foreign body—stenosis may result from the organization of granulation tissue or to the after effects of perichondritis.

3. Trauma may bring about a wide variety of stenosis. Stab wounds, injuries in automobile accidents, blows received in boxing, falls against benches, etc., are responsible for fractures, wounds and contusions that may result in obstruction unless given proper attention at the time. Stenosis following intubation may be traumatic, but the etiology is complicated by the condition for which the intubation is done.

4. Sloughing from infection or disease. Sir St. Clair Thomson states that tertiary syphilis of the larynx is perhaps the most frequent cause of cicatricial stenosis. I feel sure that this is not true in this country. I have seen only two cases. Diphtheria and streptococcic infection may result in this condition. I have seen obstruction following typhoid, but these were rather cases of collapse following loss of some of the cartilaginous framework.

5. All authorities agree that a large number of cases are due to a faulty tracheotomy in which the cricoid is severed. Jackson estimates that 85 per cent of his cases were due to this cause. He

accounts for this result on the ground that the subglottic larynx, especially in children, is lined with loose, vascular tissue which swells easily. The presence of a tracheotomy tube causes irritation and the formation of granulations which later lead to obstructing scar tissue. This is unquestionably true, but it does not seem to me a quite adequate explanation. If it were true, stenosis following intubation should be more frequent. I have a strong feeling that another element enters into the picture. The cricoid ring acts as a brace against the pull of the muscles attached to the cricoid. When it is cut, the ends are separated by the tracheotomy tube and probably pulled farther apart by the muscles. In a short time considerable distortion of the larynx results. The lower end of the cricoid bulges forward and there is a noticeable narrowing of the anteroposterior diameter. In some of these cases a fixation of the cricoarytenoid articulation on one or both sides is noticed. They show no scar tissue or evidence of inflammatory condition in the region of the arytenoids. My attention was first directed to this five years ago by a case which I shall describe later.

TREATMENT

In the management of these cases for practical purposes we may consider only three methods:

1. Gradual dilation by means of tubes or rubber core molds.

2. Laryngo-fissure followed by dilation by means of tubes or core molds.

3. Laryngo-fissure, removal of scar tissue and granulations, the application of a skin graft and later the use of tubes or core molds. In any case, it goes without saying, that if a low tracheotomy has not been done the performance of one is the first step.

The use of the first method has yielded splendid results in the hands of Jackson, Patterson and others. In my experience it has proven ideal for pure scar tissue cases, that is, cases where ulceration due to infection or the action of a corrosive chemical has resulted in a piling up of scar tissue sufficient to produce obstruction. The pressure of a tube causes rapid absorption of the scar tissue. The first case of this kind I ever handled was a good illustration of this.

January 11, 1915, C. B., a girl 14 years of age, was brought to me. Six months before, she drank some tincture of iodine. She evidently strangled and the liquid went into her larynx and trachea. There was a very severe reaction and for some time there was com-

plete aphonia. No tracheotomy was required and in time she apparently recovered, except that her voice was very hoarse. In the course of time, however, she developed dyspnea on exertion which gradually increased until she was brought to me.

Examination showed adhesion of the vocal cords for their anterior third. There was a mucopurulent discharge coming from the trachea. It was impossible to pass an infant bronchoscope below the cricoid. It was decided to do a tracheotomy and attempt gradual dilation. When the trachea was opened quite low down, the lumen was so narrow that only a No. 2 tube could be introduced. There was profuse and persistent bleeding. For several days she required constant care to relieve the aspiration of blood and secretion. The whole tube had to be removed and cleaned frequently on account of the blood. I felt sure she would develop pneumonia or lung abscess, but she did not, and in the course of a week the bleeding stopped and we found that the lumen was larger and we were able to put in increasingly larger tubes every few days. Later the synechia between the cords was cut and the use of rubber dilating tubes begun. The scar tissue was rapidly absorbed and never recurred. The adhesion between the cords recurred, however, after removal of the tubes. This was finally corrected by incision and the introduction of the largest sized O'Dwyer intubation tube, which was worn for some time. I have recently seen this lady and she has nothing to show for her experience except a tracheotomy scar and a slightly husky voice.

There are some objections to the use of rubber tubes. It is difficult to regulate the height. It is desirable to have it come above the vocal cords, but if it slips up too far it causes irritation and reaction. Again, a tube rigid enough will not accommodate itself to the variations of diameter at different levels. These disadvantages are overcome by the substitution of the Jackson core molds. These are anatomically modeled like the O'Dwyer tubes and fit into place perfectly.

In using this method, it is necessary to keep the patient under observation fairly closely. Unless the core mold is tied down to the tracheotomy tube it will often be dislodged. On the other hand, some cases, especially young children, will show so much reaction that the tube, or core mold, must be removed. The larynx is a rigid box and will not tolerate a great deal of pressure. This seems to apply particularly to post-traumatic cases.

The second method has been championed particularly by von Schmiegelow. The great advantage it has is that the larygo-fissure permits more give to the larynx and permits the use of large dilators. This, with the fastening of the tube in place with a silver wire, allows the patients to get along with less observation. The gratifying results over a long period of years reported by von Schmiegelow are worthy of serious consideration.

The third method has been endorsed by St. Clair Thomson, Negus, Arbuckle and Figi. In it the larynx is opened and examined, scar tissue and granulations are removed and a skin graft is applied and held in place by a sponge or mold of Stent material. In a week or ten days the mold is removed and the larynx closed over a rubber tube, a core mold or an intubation tube. This method is particularly adapted to cases resulting from section of the cricoid ring or from trauma with consequent distortion. I should like to offer one addition to the technic described by others. Believing, as suggested above, that the section of the cricoid ring and the separation of the cut ends results in a distortion of the larynx and some dislocation of the crico-arytenoid articulations, and that these conditions are exaggerated by the pull of the muscles attached to the cricoid, it has seemed reasonable that the ends of the ring should be dissected free and by manipulation and heavy sutures brought back into apposition. I have been gratified by the results of this procedure. Not only is a good airway obtained, but the time of recovery is remarkably short.

The following case, alluded to above, illustrates this point: J. D. B. was at work unloading large steel pipes from a flat car. One of them rolled off and struck him a glancing blow on the chest. He must have been straining at this work, as the blow caused a rupture of his trachea just below the cricoid. He immediately developed a massive emphysema extending from his forehead to his knees. He was rapidly suffocating when the surgeon reached him. His neck was enormously enlarged. In doing a tracheotomy the incision extended from his hyoid to the suprasternal notch. In opening the trachea the cricoid was cut. The patient't life was saved and in a few days the emphysema cleared up, but it was found impossible to remove the tracheal tube.

When the patient was sent to me a month later there was an almost complete subglottic closure. The vocal cords were normal in appearance, but there seemed to be a fixation of the left crico-arytenoid articulation.

A low tracheotomy was done and the larynx opened. The cut ends of the cricoid were found to be widely separated and the left one was a quarter of an inch higher than the right. The antero-posterior diameter of this part of the larynx was markedly shortened. The lower end of the cricoid seemed to bulge forward into the lumen canal. The remaining space was filled with granulations and scar tissue. This was dissected out and the denuded surface covered with a Thiersch graft on sea sponge. At the end of a week the sponge was removed and the ends of the cricoid were dissected free and brought together by manipulation and No. 2 chromic sutures. The larynx was closed over a large hard rubber intubation tube. This was removed and replaced a few times and then the triangular Jackson dilators passed occasionally to see that the passage was clear. He was able to return to work in a few weeks, but the fixation of the left arytenoid was permanent and his voice husky.

CONCLUSIONS:

1. Cicatricial stenosis of the larynx is usually amenable to appropriate treatment.

2. Dilation with Jackson rubber core molds is ideally adapted to cases of pure scar tissue stenosis. Unquestionably good results can be obtained by their use even in children in whom the cricoid has been cut if the tubes are worn sufficiently long for the larynx to develop.

3. In older children and adults, where the cricoid has been cut laryngo-fissure, skin graft and replacement of the cricoid give excellent results in a shorter time than any other method in my experience.

1304 WALKER AVENUE.

LV

BENIGN CYSTS OF THE ANTRUM*

GUERDAN HARDY, M.D.

ST. LOUIS

Cysts and mucoceles of the maxillary sinus have been but casually considered in the literature and textbooks in recent years. They are not very frequently encountered and are at times discovered only in the course of a routine radiological study of the paranasal sinuses. If small, they produce no symptoms. When large, they present a problem in differential diagnosis which is often not settled until the wall of the cyst is surgically removed and examined microscopically.

Since the terms "cyst" and "mucocele" are not synonymous, a differentiation must be made. Mucocele is a clinical rather than a pathological entity, inasmuch as the term refers to a cystic tumor lined with columnar epithelium but containing a thick mucoid or gelatinous material. A cyst, however, may be lined with either squamous or columnar epithelium, depending on its origin, and may contain a thin, watery-like secretion. Given a piece of cyst wall which has been removed from an antrum and having noted microscopically that it is lined with columnar epithelium, the pathological diagnosis is cyst of the antrum. If the contents were a gelatinous or a thick mucoid substance, it is diagnosed a mucocele. So whether it is called one or the other depends to a great extent on how broadly we use the term mucocele and also on the clinical interpretation of the contents.

Mucocele of the maxillary sinus is usually mentioned in the differential diagnosis of tumors of the antrum, but it is seldom encountered and rarely reported. Most texts mention the condition along with mucocele of the ethmoid and sphenoid sinuses. Skillern[1] even questions if true mucocele of the antrum occurs, and prefers to consider the condition as a cystic enlargement for two reasons. First, because of the fluid nature of the contents in the cases reported in the literature, and second, because of the tendency of the antral mucosa to form cysts.

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*From the Department of Otolaryngology, Washington University School of Medicine, St. Louis, Mo.

Hajek[2] states that the cysts of the maxillary sinus are usually very small and scattered throughout the membrane. Since such cysts may develop from either the gland alveoli or the gland outlets, they may be lined with either cuboidal epithelium or with columnar ciliated epithelium, respectively.

Consideration must also be given to those cysts of dental origin, i. e., follicular and radicular cysts. Such cysts are lined with squamous epithelium, and while they arise outside of the maxillary sinus, they not infrequently invaginate it. In the majority of those reported by Rosedale and Koepf[3] there was perforation of the anterior wall of the maxilla, facial deformity, and encroachment upon the antrum.

DIAGNOSIS

Cysts of the antrum should be differentiated from the nontumorous cysts of dental origin, adamantinoma, malignancies, and benign polyps. This requires a careful dental examination supplemented by dental radiography, x-rays of the sinus before and following the instillation of radiopaque fluids, careful study of the nasal cavity and lateral nasal wall, palpation of the palate, and a cytological study of the nasal secretion. Diagnostic puncture and irrigation of the antrum may give valuable information. The presence of devitalized teeth in or near the cyst should suggest a possible dental origin. Displacement of the lateral nasal wall or deformity of the palate or maxilla suggests a dental cyst or a malignancy. However, in order not to overlook a malignant growth, one should not be content until the antrum is explored and the contents removed and examined microscopically.

Originally considered as mucoceles of the maxillary sinus, the cases to be reported are now thought of as simple cysts because of the character of the contents. Clinically and pathologically they were not of dental origin. One was attached to the sinus roof, one to the lateral wall, two to the floor, and in one the site of attachment could not be determined. Two patients were edentulous and had been wearing dentures for years. None of the cysts contained thick mucoid or gelatinous material and all were lined with columnar or cuboidal epithelium.

REPORT OF CASES

CASE 1.—A. S., white, female, married, 22 years old. This patient had bronchiectasis and was referred from the chest service for an examination for foci of infection. There had been no nasal obstruction, discharge or bleeding.

Examination revealed mucopus beneath the left inferior turbinate, and chronically infected tonsils. A sinus film showed an opaque left antrum. The return wash from a diagnostic antrum puncture showed only a few shreds of mucoid matter. Another film made following the instillation of lipiodol revealed very little filling of the left antrum but good filling of the right antrum. The diagnosis was left maxillary sinusitis with thickening of the lining membrane. The blood Kahn test was negative.

Operation: Left Caldwell-Luc. A large cyst containing very thin mucoid material was found to be almost completely filling the antrum. The cyst was attached by a pedicle to the lateral wall. The remaining sinus membrane was thickened. Following removal of the mucosa of the sinus, a large naso-antral window was made. The postoperative course was uneventful.

*Pathological Study:** "The cyst wall is dense fibrous tissue. The epithelial lining where present is cuboidal. There is a plasma cell infiltration of the cyst wall and of the antral mucosa which is very edematous and polypoid."

CASE 2.—A. E., white, widow, 59 years old. This patient came to the clinic because of frequent colds and sore throats. There had been no nasal obstruction or bleeding. No teeth were present.

Nasal examination revealed nothing unusual except enlarged posterior inferior turbinate tips. An x-ray film showed an opacity of the lower half of the left antrum. The upper border of the opacity was sharply defined. Another film made after the instillation of lipiodol showed a filling defect of the floor of the same sinus. The blood Kahn test was negative.

Operation: Left Caldwell-Luc. A rounded cyst was found arising from the floor of the antrum. The remainder of the lining appeared to be normal. During removal of the cyst the wall was ruptured and about 30 cubic centimeters of clear, straw-colored serous fluid escaped. Following removal of the cyst wall a naso-antral window was made. The recovery was uneventful.

Pathological Study: "The cyst wall is dense fibrous tissue. Both sides of the party wall are lined by ciliated pseudo-stratified columnar epithelium so that the lining of the cyst cannot be distinguished from the lining of the antrum. Some chronic inflammatory changes are present."

*The pathological studies in these five cases were made by Dr. L. W. Dean, Jr.

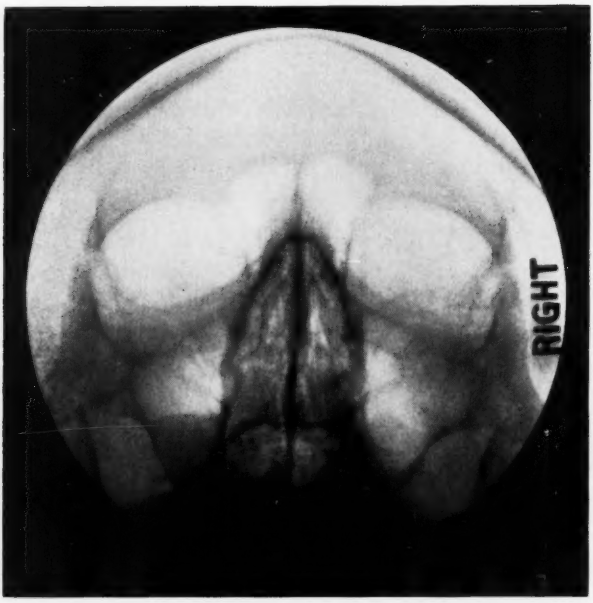


Fig. 1, Case 2. Water's position. Opacity in lower part of left antrum.

CASE 3.—C. D., white, female, married, 70 years old. This patient was seen for the first time in a surgical clinic. Her complaints were left nasal obstruction of six months duration and a swelling of the left cheek for two months. There had been no pain and no bleeding from the nose.

Examination at that time revealed a round, regular, hard, dome-shaped swelling in the region of the left maxillary antrum. The mass was palpable in the upper fornix of the buccal cavity. A diagnosis of carcinoma of the antrum was made and she was sent to radiology for deep x-ray therapy.

Six months later she was examined in the nose and throat clinic for the first time. In the interval she had received maximum dosage of deep x-ray. The swelling, while still present, had not increased in size. It was now noted that the left eye was slightly proptosed and that the external ocular movements were somewhat

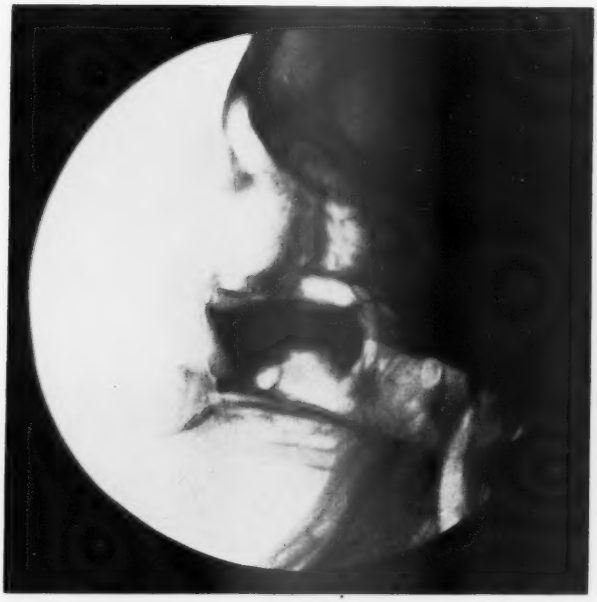


Fig. 2, Case 2. Lateral view following instillation of lipiodol into left antrum.

limited. A firm, round tumor on the face of the left maxilla could be palpated through the cheek. The cervical glands were not involved. All the teeth were out. The blood Kahn test was negative.

The left antrum was punctured and 60 to 70 cubic centimeters of thick, chocolate-colored material was aspirated. Lipiodol was instilled and an x-ray film was made. This showed a filling defect in the floor and walls of the left maxillary sinus.

Operation: Left Caldwell-Luc. The anterior face of the maxilla was found to be reduced to egg-shell thickness. A smooth moderately thick-walled cyst was found completely filling the antrum. There was no tumor mass. The posterior, orbital, palatal and medial walls were eroded. The cyst was removed and a naso-antral window was made. The postoperative course was uneventful.

Pathology Report: "A dense fibrous tissue band which appears to be a cyst wall. There is low cuboidal epithelial lining."

Fig. 3, Case 5. Water's position. Rounded shadow in floor of left maxillary sinus.

Three months later a foul discharge was noted coming from the left antrum. An x-ray film showed marked destruction of the left maxilla and erosion of the infra-orbital ridge. It was the opinion of the radiologist that there was an extensive osteomyelitis of the left maxilla. The patient was readmitted to the hospital on the plastic service, and the antrum was again explored. Most of the medial, anterior and lateral walls of the antrum were found to be necrotic. This diseased bone and the necrotic zygoma were removed. Sections of these showed no malignancy. There was only evidence of chronic inflammation.

Eight months later word was received from a niece that the patient had had no further trouble.

CASE 4.—M. J., white, female, single, 24 years old. This patient was referred from the eye clinic for an examination for foci of infection. She had a punctate keratitis.

Fig. 4, Case 5. Section through cyst wall.

Examination showed the ear, nose and throat essentially negative. A routine sinus film was taken in the Water's position. This revealed a semi-opaque structure in the upper part of the left antrum. A lateral film made some weeks later showed the mass to be larger and more irregular in outline. The return from diagnostic puncture was clear.

The general physical examination was negative except for obesity. The blood Kahn test was negative.

Operation: Left Caldwell-Luc. Attached to the roof of the antrum anteriorly was a cystic mass measuring about 4.0 cm. by 3.5 cm. in the greatest diameters. It was thin-walled and contained thin, clear, straw-colored fluid. The cyst was removed and the base cauterized. A left antro-nasal window was made and the antrum was packed with vaseline gauze. The postoperative course was uneventful.

Pathology Report: "A thin-walled cyst lined by pseudo-stratified epithelium."

CASE 5.—A. L., white, female, married, 27 years old. This patient came to the clinic complaining of frequent sore throats. Her tonsils had been removed three years prior to this visit. There had been no nasal obstruction, bleeding, or other nasal complaints.

Examination: The nose was negative except for hypertrophied turbinates. There was a small amount of adenoid tissue present. On transillumination all the sinuses appeared clear. An x-ray film of the sinuses revealed a rounded soft tissue shadow in the floor of the left maxillary sinus. Since it was thought that this might be arising from a tooth root, she was referred to dental surgery for study. It was the dentist's opinion, however, that the teeth were not involved and that the shadow was not of dental origin. The blood Kahn test was negative. The washings from diagnostic puncture of the antrum returned clear.

Operation: Left Caldwell-Luc. On removing the anterior wall of the superior maxilla, a cystic elevated area was noted on the floor of the sinus. This was filled with slightly thickened fluid. The cyst was removed but the remainder of the mucous membrane, which appeared normal, was not disturbed. A naso-antral window was made. The postoperative course was uneventful.

Pathology Report: "Sections reveal what resembles a thin, fibrous cyst wall lined by pseudo-stratified columnar epithelium. There is a plasma cell infiltration of the cyst wall."

COMMENT

It is noteworthy that the four patients having cysts in the maxillary sinus without deformity had no symptoms referable to the antrum or nose. Two came to the clinic for treatment of colds and sore throats. The other two were referred for an examination for foci of infection in the upper respiratory tract. In each instance the cyst was found in the course of a routine sinus study. In none of the cases without deformity was there any evidence of bone destruction.

SUMMARY

Five cases of cyst of the maxillary sinus are reported. The term "cyst" is used because none contained the thick, mucoid or gelatinous material which is characteristic of a mucocele.

Mucocele is a clinical diagnosis, inasmuch as it refers to any cyst containing thick, mucoid secretion and lined with cuboidal or columnar epithelium.

The surgeon should note carefully the character of the contents of a cystic mass in order to aid the pathologist in making a differentiation.

HUMBOLDT BLDG.

BIBLIOGRAPHY

1. Skillern, R.: The Accessory Sinuses of the Nose. J. B. Lippincott, Co., Philadelphia. Second edition, p. 136, 1916.

2. Hajek, M.: Nasal Accessory Sinuses. C. V. Mosby Co., St. Louis. Fifth edition, p. 178, 1926.

3. Rosedale, R. S., and Koepf, S. W.: Nontumorous Cysts of the Maxilla: Interesting Cases and Discussion. ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY, 46:652 (Sept.), 1937.

LVI

HEARING ACUITY AND STAMMERING

M. Arline Harms, M.E.

AND

J. Y. Malone, M.D.*

Milwaukee

Speech defects in pupils of elementary grades have been estimated by different investigators to be from 4 per cent to 18 per cent, which percentage would mean that at least one million children have defective speech. Thus, they outnumber the crippled, blind and deaf combined. It is estimated that 10 per cent of those with defective speech are stammerers. Inasmuch as speech is our master instrument for expression, this subject should receive far more recognition and correction than is done at present. It is impossible to estimate accurately the social, educational and economic loss as a result of these defects; but those estimates that have been made run into the millions annually.

Every human being is born speechless, but, excepting congenital defects, has a good potential speech mechanism. Thus, speech is a social habit developed through imitation. This involves not only the mental motor mechanism, but also the proper receiving mechanism. Therefore, defective hearing is a legitimate cause for defective speech, especially if this defective hearing occurs during the ages when speech habits are being formed.

In order to get a correct perception of the sounds of the English language it is necessary to have an acuity of hearing range of vibrations from one hundred to twelve thousand per second. When one speaks of defective hearing they must take into consideration not only the gross defects through the entire range, but of island defects in any portion of the vibration range.

Inasmuch as stammering is a type of speech defect, whatever principles apply to speech correction apply to stammering correction. In addition, the stammerer has some individual problems. A review of the literature reveals that no attempts have been made

*From the School of Medicine, University of Wisconsin.

to relate hearing acuity to stammering, although there is frequent reference made to the relation of the hearing acuity and certain types of speech defects. It is very apparent from the literature that the greatest toll in hearing loss is taken by childhood diseases which occur during the first seven to eight years of life. There is ample evidence to show that as the causative factors of deafness are removed hearing acuity may improve (Crowe[1]). Thus, the greatest good can be accomplished during these years. It is our opinion that hearing acuity should be tested on all school children by the end of their first-grade year, and that these tests are just as important as visual acuity tests. This is the time when existing pathology can be removed to the greatest good. It is also felt that this testing should be done with audiometers, as investigators have found that parent's, school teacher's and nurse's observations have missed the majority of these hard-of-hearing children. Newhart[2] and others have shown that in schools where audiometer testing has been used they have been able to reduce appreciably the percentage of retarded students, repeating students, etc., by removing existing pathology or placing these children in special classes.

Our object in this study has been to evaluate the possible relationship between hearing acuity and stammering. This thought was stimulated by a case having normal speech and hearing, developing a hearing loss which was followed by stammering.

REPORT OF CASES

CASE 1.—Zoe, age 13, learned her letters and many unusual words up to the age of four. Speech and hearing were in every way normal at this time. At the age of four she had scarlet fever complicated by quinzy on the left side. Following this illness she had difficulty in spelling and in carrying a tune; in fact, she was punished several times in school for being inattentive. At the age of twelve, "A more intelligent and thoughtful teacher fell to my lot, and she found that my inattention was due to lack of hearing. This was reported to my parents, and I was immediately put under medical treatment for eustachian tube blocking. After a year's treatment my hearing was normal in my right ear and very good in my left ear. My inability to spell and remember correct pronunciation has greatly improved, and with that improvement the hesitancy in my speech which occurred from confusion as to the correct pronunciation has largely disappeared. When I am awfully tired or under heavy emotional stress there is a tendency for a return of the speech hesitation."

With this lesson before us, a review of the literature was made which demonstrated a need for a study of this type. Our first act was to make a national survey of all the schools for the deaf and hard of hearing. This was done in the school year of 1936-37. The questionnaire was sent to these schools asking, first, if they

found any stammering among those who were totally deaf. Second, had they found any stammering among those with some percentage of hearing. If so, what percentage of hearing did they have, and what test was used to calibrate the acuity? The questionnaire was sent to 209 schools, 95 of whom answered the questions. The school population of those that answered constituted 74 per cent of the total school pupils, so that the response could be called satisfactory. From the total of 14,558 students there were found four cases of stammering who had no residual hearing. Their history was incomplete, so that we do not know whether they had some hearing at one time or whether they were congenitally deaf. At least, the survey showed that stammering among the totally deaf is negligible. Of those students having residual hearing there were 34 known cases of stammerers. In these 34 cases the hearing loss varied from 22 per cent to 80 per cent, with an average of 46 per cent for the group. Obviously their hearing loss must be great or they would not be included in these institutions. These cases had good hearing at one time.

One can conclude from the survey that stammering is rare in one who is totally deaf, and that it is much more frequent in cases with residual hearing, especially if their hearing has been better in former years.

Sixty-two consecutive cases of stammerers were tested with a 2-A audiometer, and in each case there was a hearing loss ranging from 10 per cent to 22 per cent. None of them were aware of their hearing loss. All of these stammered before eight years of age. Fifty-eight of the 62 cases gave positive histories of childhood diseases such as measles, scarlet fever, diphtheria, whooping cough and otitis media. This group of 58 cases may reasonably be assumed to have had normal hearing originally and, therefore, a start for normal speech development. When the defective hearing interrupted these normal sounds, which in turn could cause a confusion, there resulted a speech defect, namely, stammering.

Two cases were observed in which the hearing acuity was tested and found to be normal; they also had normal speech. Later these cases had diseases which caused a reduction of their hearing, and stammering developed. Even though the hearing acuity improved after prolonged treatment, the stammering persisted until the cases were speech analyzed and received speech treatment. It is not surprising that the stammering persisted, for during the years of normal hearing, certain sound pictures were formed; during the period of poor hearing, distorted sound pictures were formed; and

after the hearing improved there was a third set of sound pictures formed. These three sound pictures of the same words can conceivably produce so much confusion that the individual becomes undecided on trying to use these words, and thus the speech defect of stammering would develop and persist. Others might go through the same cycle, but upon return of their normal hearing overcome the confusion and thus overcome the stammering. The difference of the two types of individuals is probably due to the fact that the type that is able to overcome their confusion has a more stable emotional makeup.

CASE 2.—D. He began to talk when he was about two years old, and was brought in for speech examination at the age of four because he persisted with infantile speech. At this time there was no indication of hearing impairment. Shortly after this examination the child contracted chickenpox, which was followed by a "bad cold which left the child's ears running." This persisted for about four months. During the time that the ears were discharging the child began to stammer. This had persisted for two months when the child was again brought in for speech correction. Speech training and oral stimulation close to his ears was immediately instituted and continued for three months, after which time there was no longer any stammering, and to the spoken and whispered voice the hearing was normal.

Occasionally cases were found to stammer only upon words whose sound frequency fell within the frequency of greatest hearing loss. Following is a case which illustrates stammering only on words in the island defect:

CASE 3.—Mike. Normal speech and hearing were present up to the age of 3½ years, when he had scarlet fever and acute otitis media. A year later he had measles with acute otitis media. There was from 40 to 50 decibels loss of hearing in the left ear throughout the entire range; while in the right ear there was 120 decibels loss from 128 up to 1024 frequency, 1024 to 8192 the loss was between 80 and 90 decibels loss. He stammered only on words starting with sounds of low frequency. The patient was instructed to study words starting with the low frequency range, and after several months of careful observation the patient did not stammer except when he was under unusual stress or when he did not know what he wanted to say.

SUMMARY

Data has been presented to show that stammering is rare in those with a total loss of hearing. It becomes more frequent in those with approximately 50 per cent loss of hearing. In 62 consecutive cases of stammerers, who were unaware of any hearing loss, there was a loss of from 10 to 22 per cent, which strongly suggests a relationship between hearing loss and the development of stammering.

Cases are cited showing normal hearing and normal speech followed by disease producing some loss of hearing, and during the time that poor hearing was present, stammering developed. Also, a case was presented showing stammering on words falling within the island defect of hearing acuity.

CONCLUSION

Evidence is presented which suggests that loss of hearing acuity during the period of speech formation is a cause of stammering. Therefore, the hearing should be tested in these cases when one is attempting to determine the cause of the speech defect. When hearing defects are found they should be treated by removal, if possible, of any pathology causing the hearing loss. Hearing aids should be used in speech correction of these cases if the hearing cannot be improved to a useful level.

324 East Wisconsin Ave.

BIBLIOGRAPHY

1. Crowe, S. J., and Baylor, John W.: The Prevention of Deafness. J. A. M. A., 112:585 (Feb. 18), 1939.

2. Newhart, Horace: The Early Detection and Treatment of Defective Hearing in Children. J. A. M. A., 109:1620 (Nov. 13), 1937.

LVII

LATENT PERIOD OF THE CROSSED STAPEDIUS REFLEX IN MAN*

H. B. PERLMAN, M.D.

AND

T. J. CASE, M.D.

CHICAGO

A great deal of our present knowledge of the way the nervous system works is based on studies pertaining to the neurophysiology of an isolated unit of reflex activity in the nervous system, particularly the spinal reflex, and these have been a guide to our understanding of the acoustic reflexes of the middle ear muscles as well as to the general behavior of the central cochlear nuclei. One phase of this study on the spinal reflex has been the determination of the latency period and the breaking down of this figure by appropriate experiments into a number of component parts such as the conduction time along the efferent nerve, latency of receptors, central latency and conduction along the efferent nerve for a spinal flexor response, i. e., the knee jerk. The accuracy of the time determinations made by successive experimentors has been limited by the apparatus that was available to them at the time. The refinement of apparatus for stimulating and recording nervous and muscle activity has progressed rapidly and has permitted an extension of experimental procedures pertaining to function of the nerve fiber and its receptors (Gasser and Erlanger,[1] Adrian,[2] etc.). In 1906, Sherrington[3] studied the flexor reflex response in the spinal cat and determined the latency with the use of a myograph and related it to intensity, frequency and duration of the stimulus. He found the latency to be in the neighborhood of 26 msec. (milliseconds). In 1911, Jolly[4] studied this problem with the knee jerk, using the muscle action current as an index of muscle activity. In the decapitate cat he found a latency of 7.5 msec., which he broke down into a number of components, such as the latency of the afferent endings, 0.9 msec.; afferent nerve conduction, 1.5 msec.; motor endings

*From the Division of Otolaryngology of the Department of Surgery, and from the Otho S. A. Sprague Memorial Institute, the University of Chicago.

and motor action current, 2.8 msec., leaving 2.3 msec. as the synapse time in the spinal cord.

In 1915, Forbes and Gregg[5] studied the spinal flexor response, also using the muscle action current as indicator of activity instead of the relatively slow myographic methods, found a latency of 8.1 msec., and determined the components due to the time of conduction along the afferent and efferent nerves.

Similarly, by recording motor nerve action currents, Eccles and Sherrington[6] in 1931 studied the latency of the flexor reflex in the spinal cat and obtained 10.4 msec. as the total latent period, which was broken down into afferent nerve conduction 3.8 msec., efferent nerve conduction 2.4 msec., and central reflex time of 4.35 msec. They found that increasing the stimulus strength would reduce the latency by 2 msec., and concluded that this reduction was limited to the central reflex.

While various reflex muscle contractions to sound stimuli in decerebrate animals have been known for many years (Goltz,[7] 1892) and have been recorded as action currents (Forbes and Sherrington,[8] 1914), the latency between the onset of sound and muscle contractions in man has not been previously determined.

Kato,[9] in his classical researches on the middle ear muscles in 1913, reinvestigated the long known response of the middle ear muscles to sound stimuli and determined the latency of this acoustic reflex for the tensor tympani in the rabbit. He used a myograph for recording muscle contraction, and a mirror attached to a pneumatic system to record the onset of the sound stimulus. With this relatively crude apparatus he found the latency of the tensor tympani to a loud stimulus to be about 20 msec.

Similar observations on both the tensor and stapedius were made in 1929 by Kobrak,[10] who found a latency of .0725 second for the stapedius and .109 for the tensor tympani. Recording labyrinth pressure changes by means of a capillary tube, Tsukamoto[25] measured the time between sound onset and the rise of labyrinth pressure resulting from the associated contraction of the middle ear muscles. He obtained an average latency of .37 sec.

No further observations on the latency of the middle ear muscle contractions to acoustic stimuli were made until 1933[11] and 1935,[12] when Lorente de Nó reported on extensive studies which correlated closely the gradually growing information on the spinal reflex response with findings of the behavior of the middle ear mus-

cles of the rabbit. The reflex arc involved in the contraction of the middle ear muscles probably consists of the spiral ganglion, central cochlear nucleus, superior olive complex, motor cells for the stapedius muscle, and motor end plate in the stapedius muscle. Lorente de Nó's fundamental studies emphasized the importance of integrating the growing knowledge of neurophysiology with the function of the cochlear nerve as determined by the behavior of the middle ear muscle reflexes. He used sensitive myographs to record the movement of the tensor tympani muscle. With the apparatus he had available, he stated that he was unsuccessful in recording the muscle action currents. However, his observations showed clearly the similarity in the properties of the acoustic tensor reflex with the flexor spinal reflex and pointed to rhythmic processes in the cochlear nuclei initiated by a sound stimulus and recurring about 20 times a second. He made studies of the relation between latency and duration and intensity of the sound stimulus and found the latency of the tensor reflex to intense tones to be 10 to 12 msec., its contraction reaching a peak in about 100 msec. Using the observations of Davis and Saul[13] on the time factors involved in the conduction of action currents along the auditory nerve to the geniculate body, 7.8 msec., probable conduction time in the motor nerve, 3 or 4 msec., he calculated the central latency to be 4 to 6 msec. Recently, Hallpike[14] determined the latency of the middle ear muscle contraction in the cat to acoustic stimuli by noting the time when change in the magnitude of the cochlear potentials occurs due to the dampening of the ossicular chain as a result of contraction of the middle ear muscles. He found that about 10 msec. elapsed between the onset of the sound and the first noticeable decline in the amplitude of the cochlear potentials.

The observations which we wish to record here are on the human stapedius muscle. Its contraction to acoustic stimuli was first observed in 1929 by Lüscher.[15] Further observations in human beings have been made by Kobrak,[16] Lindsay, Kobrak and Perlman,[17] Potter[18] and by Perlman.[19] These observations have firmly established the protective function of the stapedius muscle, its response to sound stimuli at a certain level (65 d.b.) above threshold, the absense of response below this level, its response to bone conducted sounds as well as to air conducted sounds, its response to sound entering the opposite ear as well as the same ear. They have likewise established that sounds more than 65 d.b. above hearing threshold are louder in an ear unprotected by an active stapedius muscle, as in certain types of facial paralysis, than in the opposite

normal ear. The acoustic contractions of the human tensor tympani muscle have not as yet been observed, though one of us (H. B. P.) has devised a method similar to that suggested by Blake and Bing[20] for visualizing the tendon in man in the anterior fold of Troeltsch. The determination of the latency of the stapedius reflex response has been delayed because of the inaccessibility of the muscle to the myograph. An attempt was therefore made to detect the muscle action currents of the stapedius muscle. This was successful. We have so far been unsuccessful in obtaining action currents from the tension tympani muscle. The latency period of the middle ear muscles is not only of interest as a neurophysiologic phenomenon but is of clinical importance in the question of acoustic trauma, both in the experimental animal and in man, since during this latent period the perceptive mechanism is unprotected. Thus it has been shown[21] that the animals exposed to frequently repeated loud sound stimuli show evidence of damage to the perceptive mechanism as tested by the conditioned reflex, sooner than do similar animals exposed to a continuous though equally loud sound stimulus. With repeated stimuli there is a repetition of latency periods during which the acoustic oscillations of the conducting mechanism are undampened or unimpeded, thus they more violently stimulate the end-organ. Comparable situations occur in industry for operators of pneumatic hammers as in riveting, and for operators of metal stamping machines, while operators of rapid fire arms are exposed to similar acoustic trauma in military activities. Furthermore, one of us[19] has shown that the ear unprotected by the stapedius muscle fatigues much more severely to loud sound stimuli than the ear which is so protected.

A number of patients were investigated and several satisfactory records were obtained in four patients. In Case 1, the patient D. W., a boy, aged 13, showed the tendon of the stapedius and the stapes lying freely exposed in a radical mastoid cavity. Many previous observations showed a very brisk acoustic reflex contraction. In many of the patients who present a visible stapedius reflex an adherent atrophic scarred drum or a similar tissue covers the tendon and stapes. This may be sufficient to insulate the minute muscle action currents generated by the contracting muscle from the electrode and may be one reason for our failure to obtain records on a number of patients. In this patient, as in the others to be reported, a small cotton wool ball soaked in Ringer's solution and attached to a fine insulated wire leading out of the ear, was placed directly on the tendon of the stapedius muscle as illustrated in Fig. 1. The other electrode was a silver disc pasted to the skin

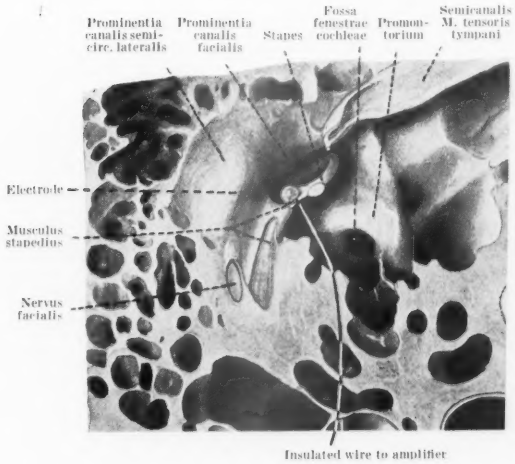


Fig. 1. Illustration (after Tandler) of stapedius muscle and adjacent structures. The placement of the electrode on the stapedius tendon and lead to amplifier is represented.

over the mastoid with electrode jelly. The more direct pickup by the application of the two electrodes directly within the muscle itself was, of course, impossible. The currents generated were led by these wires to suitable amplifiers and then to a moving loop oscillograph. The record of the acoustic stimulus was made by placing a microphone at the end of the sound tube at the patient's good ear and recording through another amplifier on another mirror of the oscillograph. The third wave train seen on the records is a 1000 cycle timing wave. Since the acoustic stimulus was a 1000 cycle tone, the two lower wave trains on the record have a similar wave form, the difference being in the time and shape of the sound onset. The acoustic stimulus was produced by an audio-oscillator with suitable amplification and conducted from the loud speaker through a heavy garden hose to within 2 cms. of the external auditory meatus of the normal hearing or opposite ear. The sound was started by a switch controlled by the driving mechanism of the oscillograph. The total elapsed time of exposure on the recording

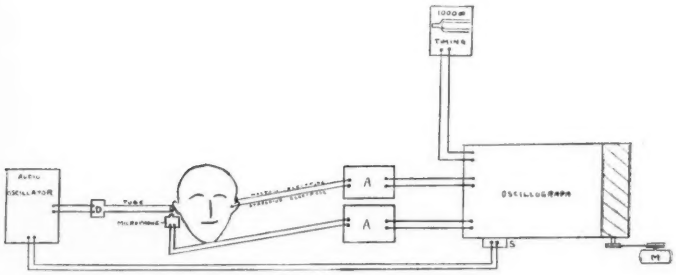


Fig. 2. Diagram representing arrangement for studying latency of the acoustic reflex of the stapedius muscle in man. *A* represents amplifiers; *D*, loudspeaker for sound output; *M*, motor driving film in oscillograph; *S*, switch synchronizing sound onset with oscillograph recording.

film was about one-twentieth of a second. The general setup of the experiment is illustrated in Fig. 2. A number of records were obtained and a representative one is illustrated in Fig. 3. The record chosen for illustration was particularly interesting because it contains two superimposed runs—one when the audio-oscillator failed to go on and the other with the sound stimulus. It is easy to recognize the changes occurring with the contraction of the stapedius muscle when comparing this track with the one below showing the base line undisturbed due to the absence of the acoustic stimulus. The beginning of the stapedius muscle action current is indicated by an arrow. The onset of the sound is almost rectangular. The rectangular onset of acoustic pressure is a very difficult physical achievement. The record shows that only about one msec. elapses between the appearance of the first wave and that of the sustained full strength wave front. We therefore measure the latency from the onset of the tone to the onset of the action current, and find this in six records to be on the average 11.4 msec. (See Table 1.)

CASE 2.—D. C., age 15, has been under observation in the clinics for chronic otitis media, left. The drum is largely destroyed, exposing the stapes head without the articulating arm of the incus. The stapes moved briskly when sharp, loud sound stimuli were produced at the opposite ear. The opposite ear functions well with a

TABLE 1

Determinations on six records obtained on February 17, 1939, from D. W.

Latency in msec.	12.5
	11.0
	11.5
	10.4
	11.0
	12.1
Average	11.4

TABLE 2

D. C., March 11, 1939

Latency in msec.

10.0	Record 1
10.8	" 2
10.5	" 3
10.1	" 4

TABLE 3

D. C., March 18, 1939

Latency in msec.

10.7	Record 1
9.75	" 2
10.00	" 3
10.5	" 4
9.00	" 5
9.5	" 6
9.05	" 7
9.85	Average

TABLE 4

Mr. C., March 17, 1939

Latency in msec.

10.8	Record 1
9.8	" 2
10.0	" 3
10.1	" 4
8.7	" 5
9.5	" 6
9.8	Average

TABLE 5

Mr. C., March 31, 1939

Latency in msec.

11.5	Record 1
10.5	" 2
10.5	" 3
9.3	" 4
10.4	Average

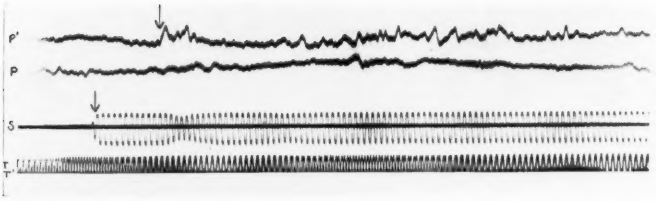


Fig. 3. Record of stapedius muscle action currents in D. W. in response to a 1000 cycle tone at about 100 decibel level. This figure is a double exposure, the first being made without the sound stimulus and the second with a 1000 cycle sound. *P* represents the electrical activity between the mastoid electrode and the electrode on the stapedius tendon when the sound stimulus was absent (Base Line). P^1 represents the activity in the presence of a sound stimulus. The onset of the sound stimulus is indicated by an arrow. The onset of the muscle action currents is also indicated by an arrow. The line extending through the sound tract represents the room noise level at the microphone pickup at the good ear when the sound was off. The 1000 cycle timing waves are superimposed in *T* and T^1.

dry central perforation. A series of four observations were made on March 11, 1939, in the manner described above, and seven records were similarly obtained on March 18, 1939.

Table 2 lists the results obtained at the first sitting and Table 3 the results obtained at the second sitting. A representative record is illustrated in Fig. 4.

CASE 3.—Mr. C., aged 36, had a dry central perforation of the right drum, exposing the incudo-stapedial articulation, the crura of the stapes in the fossa of the oval window, and the tendon of the stapedius muscle. The tendon moved briskly when loud sound stimuli were presented to the opposite ear end and, as has been observed previously in others when the incudo-stapedial joint is intact, this contraction was not accompanied by movement of the stapes but only by apparent swelling of the tendon. His hearing was good in his normal or left ear. On March 17, 1939, a series of six records were obtained in the manner described above, and on March 31, 1939, another series of four records were obtained under similar conditions. The results are tabulated in Tables 4 and 5 and representative records are illustrated in Figs. 5 and 6. These records are more striking than those from other patients, showing greater

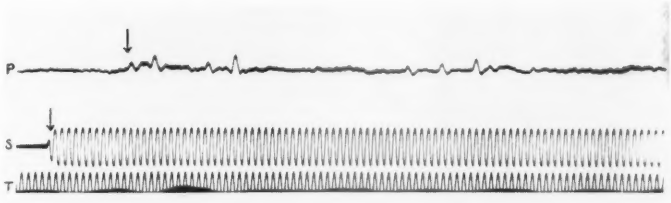


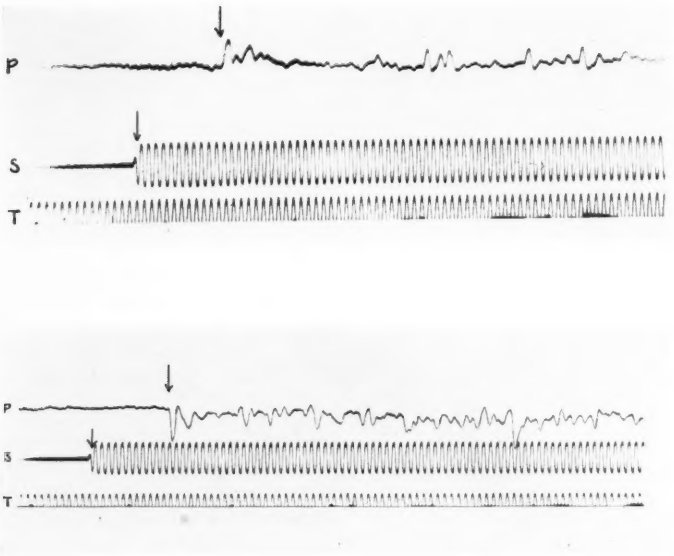
Fig. 4. Record of stapedius activity in D. C. for the left ear in response to a 1000 cycle tone at about 100 d.b. introduced into the right ear. *T*, 1000 cycle timing wave. *S*, sound stimulus with onset indicated by arrow, at *P* the electrical activity in the stapedius muscle. The first disturbance seen is indicated by an arrow. From this point the disturbance continues intermittently.

and more continuous potentials. One wonders whether this may be related to the intact incudo-stapedial articulation, and the more normal tonus of the stapedius muscle.

Two records obtained on March 31, 1939, were for a sound exposure of about one-half second. These records were made in an attempt to visualize any periodicity in action potentials that would correspond to the waves seen by Lorente de Nó in his myographs with a frequency of about 25 per second. Our records showed no definite rhythm in the action potentials, which continued throughout the duration of the exposure. (See Fig. 7.)

Case 4.—Miss J. C., aged 15, had a briskly contracting stapedius in a radical mastoid cavity on the right in response to sound stimuli introduced in the opposite ear. The stapes moved inward and backward, since the incus was absent. The opposite or left ear functioned normally. A series of three determinations were obtained on March 27, 1939, in a manner similar to that in the other experiments, with an average latency of 11.7 msec. A representative record is illustrated in Fig. 8. The average latency of the stapedius acoustic reflex for 26 determinations in these four individuals is found to be 10.5 msec.

This time factor can be examined in the light of a number of determinations in the experimental animal on the latency of the



Figs. 5 and 6. Records showing latency of the acoustic stapedius reflex contraction in the right ear of Mr. C. At *T* a 1000 cycle timing wave. At S the sound stimulus coming from an open tube 2 cm. from the left external auditory meatus, and beginning at the point indicated by arrow. At *P* the physiological track showing the abrupt onset and sustained character of the action potentials. An arrow indicates the onset of these potentials. The latency period is the time interval between the sound onset (see arrow) and the onset of the action potentials (see arrow).

integral parts of the acoustic reflex arc. The conduction time along the auditory pathways from the cochlea to the contralateral auditory tract in the midbrain is 2 msec. (Hallpike[22]). Lorente de Nó suggests about 5 msec. for conduction time in nerve fibers, calculating 200 mm. of nerve fiber and a speed of conduction of 40 meters per second, about .7 msec. for delay at the end-plate of the motor nerve in the stapedius muscle, leaving about 4.8 msec. for synaptic delays. Considering each synaptic delay to be about .7 msec., some six synapses appear to be involved. It is possible that more than one synapse is involved in the transmission of these impulses through the primary acoustic nuclei.*

*Personal communication.

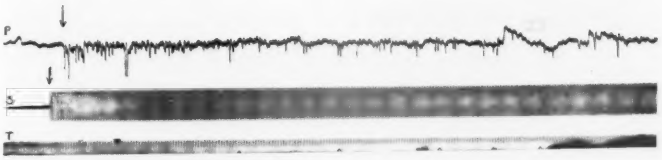


Fig. 7. Like Figs. 5 and 6, showing 1000 cycle timing wave, 1000 cycle sound stimulus, and action potentials in the opposite stapedius muscle. This record has been taken more slowly and shows better the continuous character of the action potentials.

On the other hand, Kemp, Coppée and Robinson[21] determined in the cat under moderate avertin anesthesia the latency of the auditory nerve impulses to clicks and found that responses from the trapezoid fibers appear 1.3 to 2.0 msec. after corresponding action potentials in the eighth nerve fibers, while responses from the fibers of the lateral lemniscus appear about 2.3 to 3.1 msec. after eighth nerve potentials. When conduction time was subtracted, they concluded that there was a synaptic delay of .8 msec. in the cochlear nuclei (first synapse) and .8 msec. in the superior olivary complex (second synapse). Responses from the medial geniculate appeared 4 to 5 msec. later than the corresponding wave in the eighth nerve.

While the latency of the cochlear response is .1 msec. (Davis[23]), that of the nerve action potentials as determined at the round window is .7 msec. (Hallpike[22]). The time of sound conduction from the external auditory meatus to the cochlea need not be considered, since in our experiment it was less than .1 msec. This figure of 10.5 msec. is very similar to that obtained by Lorente de Nó for the tensor tympani in the rabbit and to that obtained by Hallpike on the cat by a different experimental method but considerably smaller than the figures obtained by earlier observers with less rapid recording devices.

CONCLUSIONS

The reflex contractions of the stapedius muscle to sudden loud sound stimuli protect the cochlear structures from damage by diminishing the amplitude of vibration of the stapes. This reflex contraction is initiated in response to a certain number of nervous

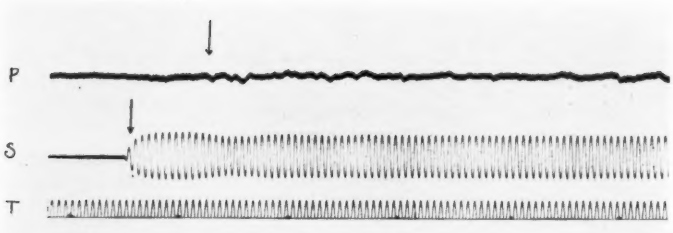


Fig. 8. Record of activity in the right stapedius of Miss J. C. in response to a 1000 cycle tone introduced into the left ear. At *T* is a 1000 cycle timing wave. At S the 1000 cycle sound stimulus, the onset of which is indicated by an arrow. At *P* the electrical activity under the electrode on the right stapedius tendon, the onset of which is indicated by an arrow.

impulses arising in the cochlea and transmitted along the reflex pathway. During the time needed for these disturbances to complete the reflex arc from sound onset to the onset of muscle contraction the cochlear mechanism is completely unprotected. This latent period is here measured for the first time in animals or man by recording the action potentials of the contracting stapedius muscle. The latency of the human stapedius muscle reflex for a loud tone of 1000 cycles is about 10.5 msec. Attention is called to the importance of this latent period in explaining the increased degree of acoustic trauma developing in the presence of rapidly repeated loud sounds as in the experimental animal and as in operators of pneumatic hammers, metal stamping machines and rapid fire arms and other similar instruments.

950 East 59th St.

REFERENCES

1. Erlanger, J., and Gasser, H. S.: Electrical Signs of Nervous Activity. University of Pennsylvania Press, Philadelphia, 1937.

2. Adrian, E. D.: The Mechanism of Nervous Action. University of Pennsylvania Press, Philadelphia, 1932.

3. Sherrington, C. S.: The Integrative Action of the Nervous System. Yale University Press, pp. 18-26, 1906.

4. Jolly, W. A.: On the Time Relations of the Knee-jerk and Simple Reflexes. Quart. J. Exper. Physiol., 4:67, 1911.

5. Forbes, A., and Gregg, A.: Electrical Studies in Mammalian Reflexes. Am. J. Physiol., 37:118-176 and 138-144, 1915.

6. Eccles, J. C., and Sherrington, C. S.: Studies on the Flexor Reflex. Proc. Roy. Soc., 107 B, 511-534, 1931.

7. Goltz, Fr.: Der Hund ohne Grosshirn. Arch. f. d. Ges. Physiol., 51:570, 1892.

8. Forbes, A., and Sherrington, C. S.: Acoustic Reflexes in the Decerebrate Cat. Am. J. Physiol., 35:367-376, 1914.

9. Kato, T.: Zur Physiologie der Binnenmuskeln des Ohres. Arch. f. d. ges. Physiol., 150:582, 1913.

10. Kobrak, H.: Zur Physiologie der Binnenmuskeln des Ohres. Passow-Schaefer Beitr. z. prakt. u. Theoret. Hals, Nasen. u. Ohrenh., 28:138, 1930.

11. Lorente de Nó, R.: The Reflex Contractions of the Muscles of the Middle Ear as a Hearing Test in Experimental Animals. T. Am. Laryng., Rhin. and Otol. Soc., 1933.

12. Lorente de Nó, R.: The Function of the Central Acoustic Nuclei Examined by Means of the Acoustic Reflexes. Laryngoscope, 45:573-595, 1935.

13. Davis, H., and Saul, L. G.: Electrical Phenomena in the Auditory Mechanism. Trans. Am. Otol. Soc., 22:141, 1932.

14. Hallpike, C. S.: On the Function of the Tympanic Muscles. Proc. Roy. Soc., 28:226-231, 1934.

15. Lüscher, E.: Die Funktion des M. Stapedium. Zeitsch. f. Hals, Nas, Ohren., 23:105, 1929.

16. Kobrak, H.: Uber die Klinische Bedeutung der Ohrmuskeln. Zeitsch. f. Hals, Nas, Ohren., 31:370, 1932.

17. Lindsay, J. R., Kobrak, H., and Perlman, H. B.: Relation of the Stapedius Reflex to Hearing Sensation in Man. Arch. Otolaryng., 23:671-678 (June), 1936.

18. Potter, A. B.: Function of the Stapedius Muscle. ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY, 45:638-643 (Sept.), 1936.

19. Perlman, H. B.: Hyperacusis. ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY, 47:947 (Dec.), 1938.

20. Politzer, A.: Diseases of the Ear. Translated by O. Dodd, Chicago. Third Edition, pp. 386, 1894 (Blake and Bing). Lea Bros. & Co., Philadelphia.

21. Finch, G., and Culler, E.: Effects of Protracted Exposure to a Loud Tone. Science, 80:41-42 (July), 1934.

22. Hallpike, C. S., and Rawden-Smith, H. F.: The Wever and Bray Phenomenon. ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY, 46:976 (Dec.), 1937.

23. Davis, H.: The Electrical Phenomena of the Cochlea and the Auditory Nerve. J. Acoust. Soc. Amer., 6:4:205, 1935.

24. Kemp, E. H., Coppée, G. E., and Robinson, E. H.: Electrical Responses of the Brain Stem to Unilateral Auditory Stimulation. Am. J. Physiol., 120:304, 1937.

25. Tsukamoto, H.: Zur Physiologie der Binnenohrmuskeln. Zeitsch. f. Biologie., 95:146, 1934.

LVIII

SYPHILIS OF THE EAR: A HISTOPATHOLOGIC STUDY

VICTOR GOODHILL, M.D.*

LOS ANGELES

Syphilitic disease of the middle and inner ear is manifested by several definite and quite constant pathological processes. The histopathology of the temporal bone in cases of "late congenital syphilis" has received very energetic study by otologists abroad. Comparatively scant attention, however, has been paid to the pathology of the "infantile prenatal" and "late acquired" types of syphilis of the ear. Relatively little has been published in the American literature on the histopathology of aural syphilis.

The present study represents a pathological investigation of sixteen cases of syphilis of the ear, eight being acquired and eight infantile prenatal (congenital). Twenty-six temporal bones in all were studied. Of the acquired cases reliable audiograms were obtained on three patients. The specimens were from the temporal bone collection of the Otologic Research Laboratory, Oscar Johnson Institute, Washington University School of Medicine. Of the 185 cases in the collection studied histologically these 16 syphilitic cases represent 8.6 per cent of the total number.

REVIEW OF LITERATURE

Baratoux (1887), is quoted by Schwartze as having described an obliterative endarteritis of the labyrinthine vessels in cases of both congenital and acquired lues.

Schwartze (1892) mentions the relative frequency with which chancres, condylomata and gummata were found at the pharyngeal ostium of the eustachian tube. This was ascribed to the use of unsterilized eustachian catheters in ear clinics. Gummata in the external auditory canal were also described.

In 1897, Walker Downie reported the case of a 17-year-old congenital syphilitic who had been completely deaf since the age of 11 years. He died of a cerebral gumma, and macroscopic observa-

*From the Oscar Johnson Institute, Washington University Medical School, St. Louis, Mo.

tion of his temporal bones showed bony obliteration of the semicircular canals, and bony distortion of the labyrinth. This was probably the first accurate description of luetic periostitis and osteitis of the labyrinth, even though the observations were macroscopic.

In 1906, Brühl reported the histologic findings in the temporal bones of a 44-year-old tabetic who had evidence of nerve deafness for five years before death. There was a complete atrophy of the nerve cells in the spiral ganglion and the fibers in Rosenthal's canal. Round cell infiltration was present in the internal auditory meatus and modiolar region.

The successful demonstration of spirochetes by Levaditi silver impregnation in the temporal bone of a congenital luetic fetus was first reported in 1911 by Grünberg. He showed that the prevailing sites of localization of the spirochetes were the blood vessels and nerves. Spirochetes were found within the lumina of vessels as well as in perivascular areas. Most of the nerve tissue was heavily infiltrated with the organisms.

Fraser and Muir, in 1917, presented an exhaustive study of the histopathology in a case of "late congenital syphilis of the ear," and summarized most of the changes which will be described later.

In 1927, G. Alexander presented his conception of the histopathology of luetic ear disease. This will be discussed more fully later.

In 1928, Schlitter reported the case of a 17-year-old congenital luetic who had been almost totally deaf since the age of seven. Microscopic study of the labyrinth revealed sero-fibrinous labyrinthitis, atrophy of the neuro-epithelial elements, and new-bone formation in the perilymphatic spaces.

Crowe, Guild and Polvogt cite thirteen cases of syphilis in their study of the pathology of high tone deafness in 1935. In only nine of these cases can syphilis be considered as a possible factor "in the causation of either the cochlear lesions or the impaired hearing."

Three distinct pathological processes in late congenital lues were differentiated in temporal bone preparations by Mayer in 1935. These were "osteomyelitis gummosa, periostitis gummosa" and "non-gummatous syphilitic periostitis productiva."

Another article on the subject by Schlittler appears in 1936. This contains an interesting review of the pathology, clinical features

and therapeutic aspects of the problem of syphilis of the inner ear, both congenital and acquired.

A monumental monograph was contributed by Otto Mayer and J. S. Fraser in 1936 on the detailed pathology of late congenital syphilitic disease of the ear. Further mention of their conclusions will be made later.

Andersen (1937) reports a case of deafness due to late congenital syphilis. The gummatous osteomyelitis and productive periostitis described by Mayer and Fraser were quite characteristic in this case also.

Additional work in pathological and clinico-pathological aspects of lues of the inner and middle ear has been reported by O. Beck, K. Wittmaack, G. Alexander, Ruskin and Hyslop, F. Christie, etc.

PRESENTATION OF CASES

A. Acquired Lues

In the eight acquired cases to be presented, positive blood serology by either Wassermann, Kahn, or both tests was confirmed in each case.

Case 1.—A. O. Autopsy No. 5150. Colored male, 49 years.

History: Patient gives history of chancre at age 22 which was treated for two weeks with "salve and powder."

Six weeks before admission, patient developed vomiting, urinary symptoms and subcutaneous masses in his neck, axillæ and groin. Upon admission to the hospital, January 6, 1933, patient had flaccid paralysis of lower extremities, bilateral facial paralysis (complete), stiff neck, bilateral Kernig and Brudzinski signs, absent abdominal and diminished deep reflexes. Mentally, however, the patient was quite clear and well oriented.

Laboratory Findings: Blood Kahn, 4 plus; Wassermann, negative. Spinal puncture, 217 lymphocytes, two polymorphonuclears. Pandy, positive; Kahn and Wassermann, negative. Gold curve, 0000133310. Total protein, 200 mgm. per cent, normal pressure.

Hearing Test: (See Fig. 1) January 9, 1933. Weber: not lateralized. Rinne: positive, right and left. Bone conduction, normal, right and left. Audiogram shows a moderate tone gap at 4096 DV for the right ear.

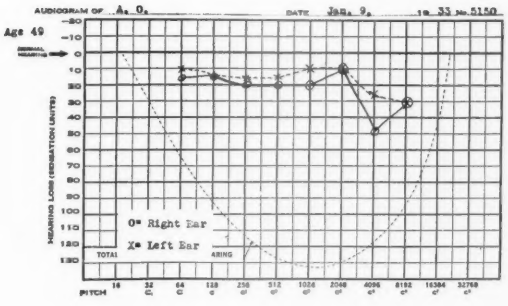


Fig. 1. Audiogram on Case 1 (5150).

Course: Patient became progressively worse, developed incontinence. Spinal fluid cell count increased to 350 per cubic mm. Temperature elevation set in and patient expired on January 17, 1933.

Autopsy Diagnoses—January 17, 1933: (1) Syphilis. (2) Lymphocytic meningitis. (3) Moderate arteriosclerosis. (4) Possible Hodgkin's disease or lymphoblastoma.

Otitic Pathology—Right Ear:

Middle Ear: Deformity of foot-plate of stapes.

Inner Ear: Small round cell infiltration throughout membranous cochlea (miliary gummata) (see Fig. 2). Marked round cell infiltration in vestibule, under maculæ of saccule and utricule, and in ampulla of posterior semicircular canal. Endolymphatic wall infiltrated also. Productive periostitis producing distortion and osseous invasion of ductus endolymphaticus (see Fig. 3).

Productive periostitis is a term applied to an apparently specific luetic bone lesion characterized by bony invasion of the lumen of any one or all of the vestibular channels. This invasion is manifested first by an irregularity in contour of the perilymphatic lumen, then by a progressive fibrosis of the perilymphatic tissue, and finally by an osseous proliferation which eventually occupies the entire perilymphatic and endolymphatic systems of the canals.

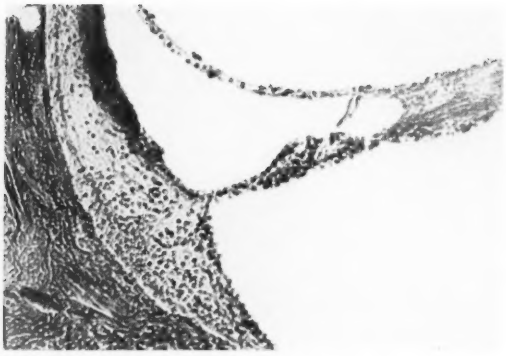


Fig. 2, Case 1. Lymphocytic infiltration in membranous cochlea. (No. 5150, Sec. 361.)

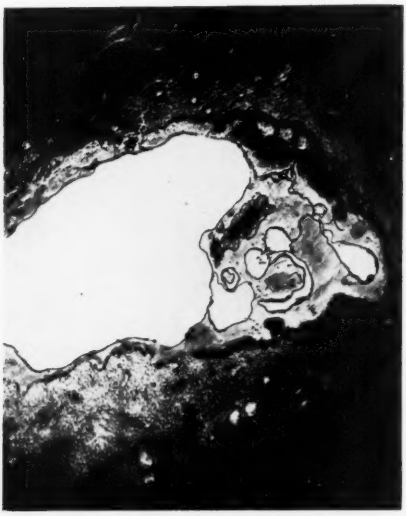


Fig. 3, Case 1. Ductus endolymphaticus with misshapen contour due to productive periostitis and invasive fibrosis. (Retouched.) (No. 5150, Sec. 532.)

CASE 2.—R. J. Autopsy No. 4486. Colored female, age 36.

History: Patient had been receiving anti-luetic therapy at Municipal Clinic during the year preceding admission. She progressively lost the use of her left arm and leg, became aphasic and disoriented. She was admitted to Barnes Hospital on March 23, 1931.

Examination: Patient in stupor with aphasia and left-sided hemiparesis. Tongue paralyzed. Unequal pupils which do not react to light. Discs hyperemic and margins blurred. Left seventh, ninth, eleventh and twelfth cranial nerves paralyzed. Flaccid paralysis of left upper and lower extremities. Absent abdominal reflexes. Bilateral Babinski positive. Hyperactive deep reflexes.

Laboratory Findings: Urinalysis: albumin 4 plus. Blood Wassermann and Kahn 4 plus. Lumbar puncture: fluid clear but under increased pressure; 21 lymphocytes. Pandy 4 plus, Wassermann and Kahn 4 plus. Colloidal gold curve 5555555540.

Clinical Diagnosis: General paresis, syphilis.

Course: Patient became incontinent rapidly, and was unable to swallow. She was discharged to her home, where she died three weeks later. Body was returned to hospital for postmortem study.

Autopsy Findings—April 11, 1931: (1) Paresis. (2) Atrophy of dorsal sensory columns of spinal cord. (3) Localized areas of cerebral cortical atrophy.

Otitic Pathology—Right Ear:

Middle Ear: Deformity and bowing of stapedial crura and footplate. Mucosa shows slight polymorphonuclear infiltration.

Inner Ear: Degeneration of organ of Corti and spiral ligament. Submucosal fibrosis of ductus endolymphaticus.

Left Ear:

Middle Ear: Purulent infection in mastoid cells. Free pus in niches of round and oval windows.

Inner Ear: Atrophy of organ of Corti and spiral ligament. Degeneration of Reissner's membrane. Productive periostitis of semicircular canals. Arteriosclerosis. Psammomata in internal auditory meatus.

Note: Varying degrees of productive periostitis are present. The very earliest manifestation is an irregularity of the bony mar-

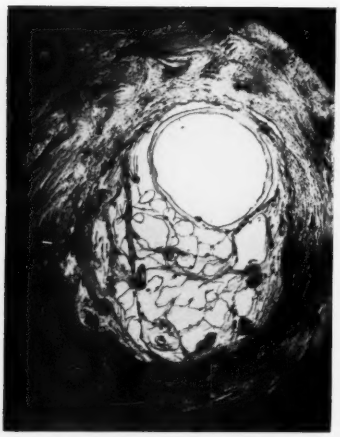


Fig. 4, Case 2. Medial limb of superior semicircular canal, showing early fibrosis and bony irregularity of perilymphatic lumen. (No. 4486, Sec. 330.)

Fig. 5, Case 2. Same case as Fig. 4. Canal here shows a more advanced type of fibrosis. (No. 4486, Sec. 261.)

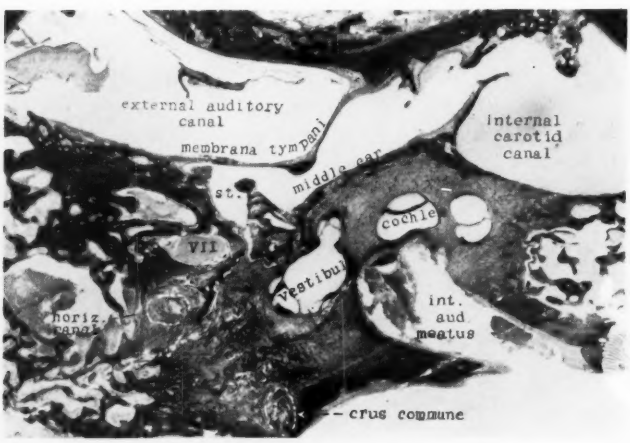


Fig. 6, Case 3. Horizontal section. Note complete cast of new bone filling crus commune, horizontal canal and part of vestibule. Suppurative labyrinthitis. Marked deformity of stapes. (No. 42-50, Lft. Sec. 373.)

gin of the perilymphatic channel of the semicircular canal. The next step in the pathological process is a progressive fibrosis of the perilymphatic tissue (Fig. 4). One sees a network of fibrous tissue surrounding collagen bodies; and as the older areas of the disease are approached the fibrous reaction becomes denser and denser until finally a mass of heavy scar tissue is the only thing seen in place of the former perilymphatic lumen. The endolymphatic lumen remains surprisingly intact in contour, being relatively unencroached by the encircling fibrosis (Fig. 5). Ultimately, new bone formation progresses in the fibrosed area until the entire canal has been obliterated.

CASE 3.—J. W. Autopsy No. 42-50. White male, age 61.

History: Two days before admission to the hospital the patient complained of mild left otalgia for several hours; the pain disappeared. Following this he felt "under par" and was drowsy. He complained of deafness, and rapidly became irrational. He was admitted on September 20, 1934, in a semistuporous state, complaining of pain in the back of his head.

Examination: Temperature 40 degrees C. Marked neck rigidity. Left pupil fixed. Right pupil reacts sluggishly to light. Right drum membrane normal. Left drum dull, rather full, peripherally injected, redness along manubrium. Abdominal reflexes absent. Diminished deep reflexes. No pathological reflexes, except for a questionable Brudzinski bilaterally. Fundi negative.

Laboratory Findings: Spinal puncture: Pressure 330 mm. H_2O. Fluid cloudy; 7,000 cells, 90 per cent polymorphonuclears. Streptococci seen on smear (positive streptococcus culture). Blood Kahn positive.

Course: The left drum membrane was incised and pus obtained. Patient became rapidly worse and expired 36 hours after admission on September 21, 1934.

Clinical Impression: (1) Streptococcic meningitis, secondary to left O. M. P. A.

Autopsy Findings: (1) Acute streptococcic meningitis; (2) acute purulent labyrinthitis (left); (3) acute otitis media (left); (4) syphilis.

Otitic Pathology—Right Ear:

Middle Ear: Essentially negative.

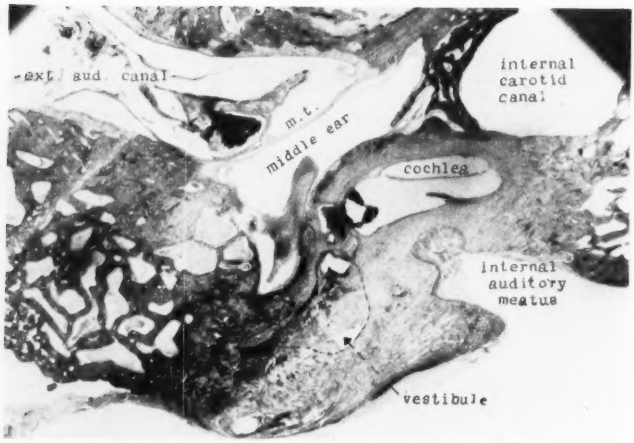


Fig. 7, Case 3. Horizontal section at level of basal turn of cochlea. Purulent invasion of labyrinth occurs via round window. New bone is seen in vestibule here also. (No. 42-50, Lft. Sec. 465.)

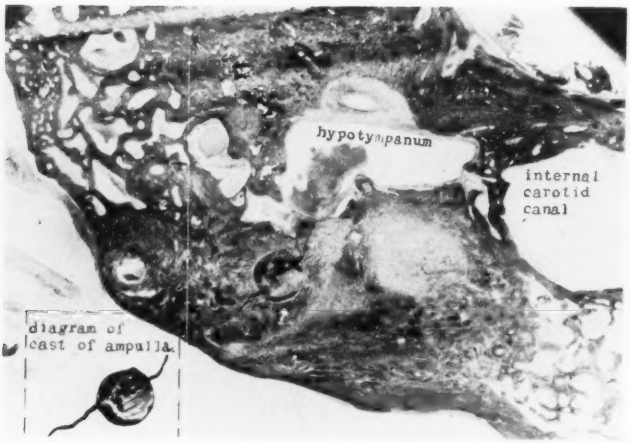


Fig. 8, Case 3. Hypotympanum. Completely ossified ampulla of posterior semicircular canal, causing fracture of surrounding otic capsule. (No. 42-50, Lft. Sec. 521).

Inner Ear: Atrophy of organ of Corti and Reissner's membrane. Pus in spiral ganglion region (entering from internal auditory meatus). Mild productive periostitic reaction.

Left Ear:

Middle Ear: Membrana tympani: Sloughed off mucosal and epidermal layers, small perforation. Purulent invasion of labyrinth via round window is clearly evident, with total destruction of round window membrane. Purulent mastoiditis.

Inner Ear: Purulent labyrinthitis with destruction of organ of Corti and Reissner's membrane. Semicircular canals and vestibule are almost completely filled by marked invasion of new bone (Figs. 6 to 9, inclusive).

The productive periostitis in this case has produced the most marked changes seen in the entire series of cases. The fibrosis has been almost entirely replaced by new bone formation (Fig. 6) and even the rather resistant endolymphatic system has been obliterated by the pathological bone.

Crus commune, ampullæ and limbs of the semicircular canals are solidly filled with bone. Fracture lines can be seen through the otic capsule as a probable result of the expansive pressure from the new bone growth within the ampulla of the posterior canal. The fracture extends from middle ear cavity through to the dura (see Fig. 9).

Case 4.—M. M. Autopsy No. 5134. Colored male, age 53.

History: Patient entered Barnes Hospital on December 8, 1932, complaining of epigastric pain, loss of weight, weakness and vomiting.

Examination: Poorly nourished colored male. Very large liver, distending upper abdomen and extending below umbilicus. Surface of mass was irregular and tender.

Laboratory Findings: Blood Kahn positive.

Clinical Impression: Carcinomatosis of liver.

Course: Patient became progressively worse and died three weeks following admission.

Autopsy Findings: (1) Carcinoma of stomach with metastases to liver. (2) Bronchopneumonia.

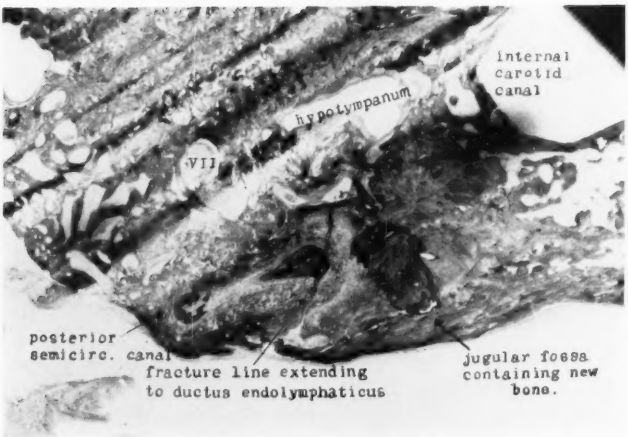


Fig. 9, Case 3. Hypotympanum. Note the two limbs of the posterior canal solidly cast in new bone, from which the fracture line diverges. The superior level of jugular fossa also contains new bone.

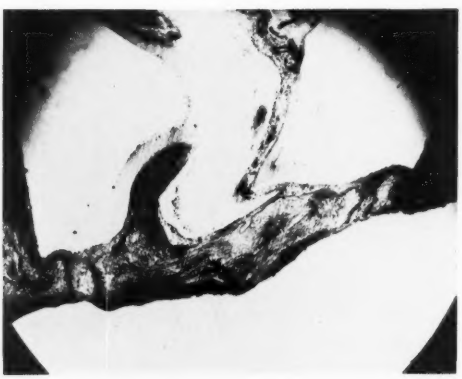


Fig. 10, Case 5. Marked deformity of crura and foot-plate of stapes. (No. 4056, Sec. 660.)

Otitic Pathology—Right Ear:

Middle Ear: Purulent otitis media. Fibrosis and pus in round window niche. Mucosal round cell infiltration. Vascular congestion. Bowing of stapedial foot-plate outward.

Inner Ear: Atrophy of organ of Corti, tectorial membrane, spiral ligament, stria vascularis, vestibular maculæ and cristæ. Stretching of Reissner's membrane due to dilatation of ductus cochlearis. Productive periostitis of semicircular canals, affecting especially the horizontal canal.

Left Ear: Not obtained.

CASE 5.—C. D. Autopsy No. 4056. Colored male, age 38.

History: Gonorrhea 10 years before admission. Wife had one still birth but no miscarriages. Four months before admission, patient developed progressive right hemiplegia following a sudden attack of unconsciousness. Ten days before admission he noticed memory changes and articulatory difficulty. Patient was admitted to Barnes Hospital on March 7, 1930.

Examination: Complete right hemiplegia, paralysis of right facial nerve, and right side of tongue. Complete right hypesthesia. Taste absent on the posterior one-third of tongue. Abdominal reflexes absent. Hyperactive deep reflexes especially on right. Positive right Babinski. Ears essentially negative. Hears watch two feet either side. Weber not lateralized. Rinné positive, bilateral.

Laboratory Findings: Blood Wassermann and Kahn both 4 plus. Spinal fluid clear; negative Kahn and Wassermann. Colloidal gold 001110000001.

Clinical Diagnosis: (1) Progressive right hemiplegia. (2) Meningo-vascular lues.

Course: Patient aspirated coffee ground vomitus, developed a terminal pneumonia and died March 23, 1930.

Autopsy Diagnoses: (1) Cystic glioma of left parietal lobe (spongioblastoma multiforme). (2) Meningeal thickening, syphilis. (3) Aspiration pneumonia with abscess formation.

Otitic Pathology—Right Ear:

Middle Ear: Increased fibrous tissue in mucosal lining. Marked deformity of crura and foot-plate of stapes (Fig. 10, Case 5). Perineural edema around facial nerve.

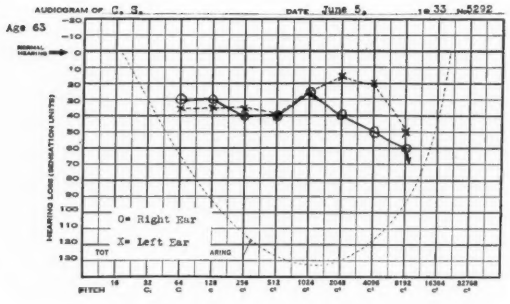


Fig. 11. Audiogram on Case 6 (5292).

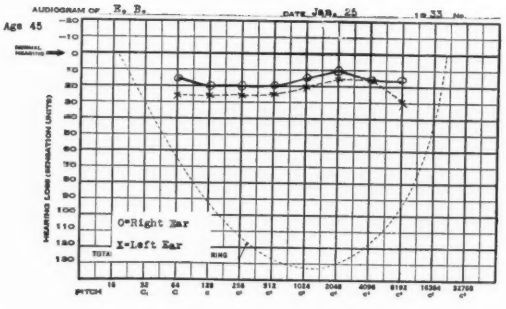


Fig. 12. Audiogram on Case 8 (5204).

Inner Ear: Terminal arteriolar sclerosis (see Baratoux). Degeneration of ganglion cells in basal turn of cochlea, vestibular end-organs and stria vascularis. Productive periostitis affecting all semicircular canals.

Left Ear: Essentially same changes as in right.

CASE 6.—C. S. Autopsy No. 5292. Colored male, age 63.

History: Patient admitted to Barnes Hospital on June 3, 1933, with complaints of difficulty in swallowing, vomiting, hoarseness, mass in neck, loss of weight and tinnitus.

Examination: Hard nodes in neck, generalized emaciation. Sluggish pupillary reactions. Both tympanic membranes thickened and retracted with shortened light reflexes. Drumheads and mallei move with Seigle speculum. Both eustachian tubes are patent, as tested by catheterization and inflation. Paresis of left vocal cord.

Hearing Test: June 5, 1933 (see audiogram, Fig. 11). Weber not lateralized. Bilateral positive Rinné test. Bilateral decreased bone conduction (Schwabach). Moderate loss for low tones. Marked bilateral loss for high tones.

Impression: Mixed type of deafness.

Laboratory Findings: Blood Wassermann and Kahn 4 plus.

Course: Patient rapidly went down hill and died on June 10, 1933.

Autopsy Findings: (1) Carcinoma of esophagus. (2) Metastatic carcinoma of trachea. (3) Tracheo-esophageal fistula. (4) Syphilitic aortitis. (5) Bronchopneumonia.

Otitic Pathology—Right Ear:

Middle Ear: Negative.

Inner Ear: Normal spiral ganglion and organ of Corti. Productive periostitis of superior and horizontal canals.

Left Ear:

Middle Ear: Stapedial foot-plate bowed.

Inner Ear: Destruction of spiral ligament. Stria vascularis adherent to bony wall. Productive periostitis of superior canal.

CASE 7.—T. B. Autopsy No. 6937. White male, age 76.

History: Patient was admitted to Barnes Hospital upon four occasions during 1936 and 1937 for the treatment of cardiac de-

Fig. 13, Case 9. Hemorrhage into perilymphatic channel of semicircular canal. (No. 4384, Sec. 260.)

Fig. 14, Case 9. Subarcuate fossa in a prenatal case showing diapedesis due to vascular fragility in smaller vessels. (No. 4384, Sec. 560.)

Fig. 15, Case 11. Medial limb of superior semicircular canal. Note triangular shape, a distortion resulting from luetic bone disease. (No. 3873, Sec. 404.)

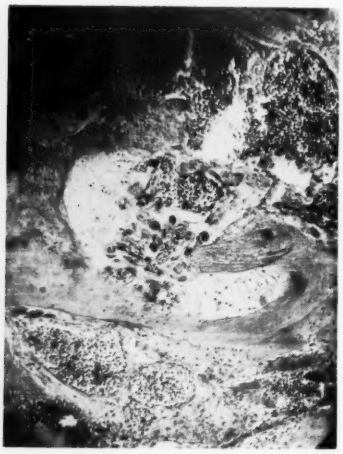


Fig. 16, Case 12. Note deficiency and malformation of globuli interossei; fibrotic bone marrow.

Fig. 17, Case 14. Marked bowing of foot-plate of stapes with surrounding lymphocytic infiltration. No. 3937, Sec. 580.)

Fig. 18, Case 14. Modiolar region of cochlea showing lymphocytic infiltration extending along basilar membrane. (No. 3937, Sec. 490.)

compensation secondary to luetic aortitis with regurgitation. He expired on July 17, 1937, of terminal bronchopneumonia.

Laboratory Findings: Blood: Wassermann and Kahn 4 plus. Spinal fluid: Wassermann 4 plus. Increased cell count. Colloidal gold curve 55555445100.

Autopsy Diagnoses: (1) Syphilitic aortitis. (2) Paresis. (3) Terminal bronchopneumonia. (4) Arteriosclerosis generalized.

Otitic Pathology—Right Ear:

Middle Ear: No marked changes.

Inner Ear: Early changes of productive periostitis producing irregular bony contours of semicircular canals. Slight perilymphatic fibrosis present in a few areas. Atrophy of spiral ganglion cells and of organ of Corti. Small round cell infiltration in Rosenthal's canal. Calcified placques in internal carotid.

Left Ear: Not obtained.

CASE 8.—E. B. Autopsy No. 5204. White male, age 45.

Past History: Malaria at age 26. Gonorrhea at 31 years. Luetic ulcer of thigh at age 42. History of bilateral otalgia without discharge at age seven.

Present History: Patient was admitted on October 31, 1932, complaining of abdominal pain and distension, hematemesis, bleeding from the gums, epistaxis and tarry stools. He had been taking large doses of morphine for pain and was somewhat addicted.

Examination: Pupils small, unequal, and do not react to light. Liver and spleen enlarged and tender.

Laboratory Findings: Blood: erythrocytes 8,600,000. Platelets 816,000. Hemoglobin (Newcomer) 105 per cent. Leucocytes 39,200. (Segmented 41 per cent, stabs 35 per cent, juveniles 6 per cent, eosinophiles 2 per cent, lymphocytes 5 per cent.)

Serology: Blood Wassermann and Kahn 4 plus.

Spinal Fluid: Kahn 2 plus. Gold curve 123343100.

Basal Metabolic Rate: Plus 25 per cent.

Hearing Test: January 25, 1933 (three months before death) (see Fig. 12). Weber not lateralized. Rinné negative on right, indeterminate on left. Bone conduction: bilaterally prolonged three seconds over that of the examiner. Lewis test: stapes not fixed on either side.

Course: Patient grew steadily weaker and expired on March 14, 1933.

Autopsy Diagnoses: (1) Syphilis. (2) Hyperplastic bone marrow. (3) Splenomegaly and hepatomegaly. (4) Arteriosclerosis.

Otitic Pathology—Right Ear:

Middle Ear: Fibrosis in mucosa of tympanic cavity, especially in niches of oval and round windows. Marked bowing of stapedial foot-plate, but no ankylosis of stapes.

Inner Ear: Productive periostitis causing irregularity in contour of the bony walls of semicircular canals and vestibular aqueduct. Edema of perilymphatic spaces. Atrophy of organ of Corti and spiral ganglion cells. A typical "herde" or focus of otosclerosis appears medial to vestibule at the level above oval window. This area can be traced in many sections and is anterior and also some-

what inferior to oval window. The bone of this "herde" is dark staining and quite vascular. There is no involvement of the stapedio-vestibular articulation by the otosclerotic focus. (Note that the Lewis test showed no stapes fixation in this case.)

Left Ear: Lesions almost identical to the right ear are present in this ear also. The otosclerotic area has the same location. Luetic productive periostitis also involves the canals here. Stapedial footplate is markedly bowed outward, but not ankylosed.

B. Prenatal (Congenital) Lues

The nomenclature in heredosyphilis has been subjected to criticism by Stokes. According to him, the term "congenital syphilis" has had wide indiscriminate use. He prefers to limit the "congenital" description more strictly to that type of syphilis which follows infection of the fetus in passing through the birth canal; meaning, in effect, that congenital syphilis is really a form of acquired syphilis. On the other hand, prenatal or heredosyphilis is a variety due to direct infection of the fetus via the placenta and the maternal and fetal blood streams. Prenatal syphilis, therefore, implies an overwhelming type of virulent infection resulting in a high fetal mortality.

The clinical features of the infant cases in this series place them all in Stokes' infantile prenatal category. The diagnosis of prenatal syphilis in the following series was based upon (a) the presence of characteristic clinical features of the disease, combined with (b) pathological demonstration of luetic changes in the viscera, and finally (c) in most cases, on positive serology in mother, child, or both. Less emphasis was placed upon serologic positivity than upon characteristic clinical and postmortem findings, since it has been demonstrated by Cooke and Jeans that syphilitic infants at birth have a negative blood Wassermann reaction in 37 per cent of cases, weak positive in 18 per cent, and positive in 45 per cent.

However, positive serology was found in four of the eight prenatal cases which follow.

Case 9.—T. B. Autopsy No. 4384. White male, age one month.

Family History: Mother has 4 plus blood Wassermann; father's serology negative. History of "bleeders" in mother's family. One stillbirth, 1923. One miscarriage, 1929.

Present History: Full term pregnancy, normal labor. Child was jaundiced at birth. Jaundice has been progressive since birth. Repeated nose-bleeds since age of two weeks. Patient admitted to St. Louis Children's Hospital on January 27, 1931.

Physical Examination: Icteric infant. Temperature 36 degrees C. Anterior fontanelle bulging, tense. Bilateral, mild epistaxis. Bleeding from palatal ulcers in mouth. Heart rate rapid. Rales at lung bases. Liver and spleen palpable. Generalized extensor spasm of upper and lower limbs. Purpuric patches on legs. Scaling of epidermis of palms and soles. Tympanic membranes appeared normal.

Clinical Diagnoses: (1) Hemorrhagic disease of the new-born with intracranial hemorrhage. (2) Congenital syphilis.

Laboratory: Blood Wassermann 4 plus. Kahn 4 plus.

Course: Infant was transfused with 60 cc. of citrated blood. This was followed by temporary cessation of bleeding. Later Cheyne-Stokes respirations developed and lumbar puncture was done. Spinal fluid pressure was increased and fluid was markedly blood stained. The following day the child suddenly stopped breathing and could not be revived.

Autopsy Diagnoses: (1) Congenital syphilis. (2) Cerebral hemorrhage. (3) Icterus neonatorum.

Otitic Pathology—Right Ear:

Middle Ear: Vascular congestion of mucosa. Bony distortion of stapedial foot-plate. Infiltration of mastoid air cells by groups of large mononuclear cells.

Inner Ear: Heavy hemorrhage in perilymphatic channels of cochlea (scalae tympani and vestibuli). Marked atrophy of organ of Corti. Heavy hemorrhage in perilymphatic regions of vestibular labyrinth (Fig. 13). Hemorrhage in spiral ganglion region. Mild productive periostitis of semicircular canals.

Left Ear:

Middle Ear: Changes similar to those in right ear.

Inner Ear: Diapedesis around veins of subarcuate fossa (Fig. 14). Organ of Corti fairly normal. Mild productive periostitis of semicircular canals.

CASE 10.—G. H. Autopsy No. 5454. Colored female, stillborn.

History: Mother was a 14-year-old negro girl, a prima-gravida at term, admitted to the St. Louis Maternity Hospital on October 11, 1933. Maculo-papular lesions were present on her genitalia and all over her body. A stillborn baby was delivered on October 14, 1933.

Serology: Blood Wassermann and Kahn 4 plus.

Autopsy (1) Stillborn. (2) Congenital syphilis.

Otitic Pathology—Right Ear:

Middle Ear: Otitis neonatorum. Marked vascular congestion.

Inner Ear: Degeneration of spiral ganglion cells and of nerve fibers. Atrophy of organ of Corti.

Otic Capsule: Liquefaction of bone marrow with intense vascular congestion. Irregularity of bony contour of superior and posterior semicircular canals.

Left Ear: Essentially the same as right.

CASE 11.—S. J. K. Autopsy No. 3873. White female, age three and a half months.

Family History: No history of lues or tuberculosis. No miscarriages.

Birth History: Three weeks premature. Forceps delivery. Very good development until three weeks before admission.

Present History: Three weeks prior to admission, child developed upper respiratory infection. Two days before admission vomited feedings. Patient was admitted to Children's Hospital on August 25, 1929. Temperature 37.8 degrees C.

Examination: Poorly nourished child. No otorrhea. Tympanic membranes appear full and moderately injected. Nasal obstruction present. Postnasal discharge of mucopus.

Chest: Dullness over left upper lobe. X-ray of chest reveals bilateral lobar pneumonia.

Laboratory Findings: Blood Wassermann 4 plus. Blood count: 41,000 leucocytes; 50 per cent neutrophiles, 50 per cent lympho-

cytes. Intradermal tuberculin negative. Spinal puncture: 56 cells, all monocytes. Pandy 1 plus. Spinal Wassermann 4 plus.

Clinical Diagnoses: (1) Syphilis, congenital. (2) Syphilis, cerebrospinal meninges. (3) Rhinopharyngitis, acute. (4) Pneumonia, secondary bronchial.

Course: Progressive dyspnea and abdominal distension occurred. Patient died five days following admission.

Autopsy: (Levaditi stain of kidney and lung revealed no spirochetes). (1) Congenital syphilis. (2) Acute otitis media. (3) Purulent bronchitis. (4) Miliary hepatic abscesses.

Otitic Pathology—Right Ear:

Middle Ear: Purulent process in antral cells. Cellular infiltration of lining mucosa. Inward bowing of foot-plate of stapes.

Inner Ear: Degeneration of cochlear ganglion cells in basal turn of cochlea. Atrophy of organ of Corti. Productive periostitis of semicircular canals, producing distortion of bony contour. The canal has become triangular in shape (see Fig. 15).

Left Ear: Not obtained.

CASE 12.—L. S. Autopsy No. 4541. Negro female, age 33 days.

History: Father and mother have both been receiving antiluetic treatment in the clinic. This is the first child. Child was born during eighth month of gestation and supposedly weighed three pounds at birth. Child had not gained weight on lactose-milk formula. Admission to St. Louis Children's Hospital on June 8, 1931.

Physical Examination: Premature, undernourished negro child, with mild serous discharge from nose and slight cervical adenopathy.

Course: Infant died five days after admission to hospital on June 12, 1931.

Autopsy Diagnosis: (1) Prematurity. (2) Pulmonary atresia. (3) Patent interventricular septum. (4) Patent ductus arteriosus. (5) Pulmonary artery thrombosis. (6) Congenital syphilis.

Note: The history of active syphilis in both parents, the prematurity of the child, and the apparent presence of "snuffles" makes a presumptive diagnosis of prenatal lues justified.

Otitic Pathology—Right Ear:

Middle Ear: Submucosal infiltration of mononuclear and eosinophilic cells. Abnormal shape of stapedial crura. Antral cells contain exudate of large giant cells and monocytes, with very few polymorphonuclears.

Inner Ear: Vascular dilatation and congestion.

Otic Capsule: Advanced ossification evident, along with abnormal degeneration of bone. There appears to be a deficiency and malformation of globuli interossei. There is early fibrosis of marrow which in some areas goes on to pathological new bone formation (Fig. 16).

Internal Auditory Meatus: Bony infiltration of perineural and perivascular spaces.

Left Ear: Essentially same changes as in right.

CASE 13.—P. B. Autopsy No. 3865. White male, age four months.

Family History: No miscarriages. Six other children living and well. No still-births.

Present History: Premature delivery (at seven months). Child normal at birth except for peeling of the skin of the palms and the soles. Following birth, infant had several convulsions accompanied by fever, cough and vomiting. One month before admission, he had an eruption over the face, body and hands. The skin eruption became progressively worse and vomiting increased. Admitted to Children's Hospital on August 15, 1929.

Examination: An acutely ill, irritable, dirty, pale, athreptic, rachitic, dehydrated baby with loose, inelastic skin covered with papules. Parietal bossing; full anterior fontanelle. Tympanic membrane red and dull. Nasal mucosa red. Lips fissured. Pharynx red. Marked cervical adenitis. Moderate rosary formation. Scattered rales over both lung fields. Distended abdomen. Enlarged spleen and liver. Temperature 39 degrees C.

Laboratory Findings: Blood Wassermann 4 plus. Kahn 4 plus. Spinal fluid Wassermann negative. Spinal fluid under increased pressure. Intradermal tuberculin negative.

Course: Infant died four days after admission, August 19, 1929.

Autopsy Diagnosis: (1) Congenital syphilis. (2) Primary bronchopneumonia. (3) Thrush.

Otitic Pathology—Right Ear:

Middle Ear: Membrana tympani: mucosa completely sloughed down to fibrous layer. Marked subepithelial cellular infiltration. Purulent otitis media.

Inner Ear: Atrophy of cochlear ganglion nerve cells and fibers. Vestibular ganglion cells appeared normal.

CASE 14.—V. L. P. Autopsy No. 3937. White female, age 10 days.

History: Infant born elsewhere, ten days before admission to Children's Hospital on October 29, 1929. Mother states that infant had jaundice at birth and bled from the mouth several days before admission. The infant was full-term, was delivered spontaneously and weighed six and one-half pounds at birth. There were no previous miscarriages. Serology of both parents unknown.

Examination: New-born female infant, 41 cm. long, respirations 56 per minute; expiratory grunt. Skin icteric. Oozing from lips and gums. Bruises all over the body. Palate icteric. Distended tympanitic abdomen. Infant expired three and one-half hours after admission to hospital.

Autopsy Diagnosis: (1) Congenital syphilis. (2) Hemorrhagic disease of the newborn.

Otitic Pathology—Left Ear:

Middle Ear: Marked bowing of stapedial foot-plate. Slight amount of purulent exudate (Fig. 17).

Inner Ear: Destruction of organ of Corti and spiral ganglion cells. Fibrinous deposit throughout labyrinth. Small round cell accumulations suggestive of miliary gummata (Fig. 18).

Otic Capsule: Productive periostitis producing a distorted cochlear bony wall.

Right Ear: Not obtained.

CASE 15.—B. J. Autopsy No. 6115. Negro male, still-born.

History: Mother is a known luetic, inadequately treated. This pregnancy was apparently normal, lasting 39 weeks. Delivery was spontaneous, without anesthetic; fetal heart sounds heard fifteen minutes before delivery. Still-born infant delivered on April 28, 1935, weighing 4,010 grams.

Autopsy Findings: (1) Thickening of skin on palms and soles. (2) Complete pulmonary atelectasis. (3) Caput succedaneum. (4) Hypertrophy of thymus and liver. (5) Congenital lues. (Microscopic sections of lung, liver, kidney, adrenal, thymus, umbilical cord, stained by the Levaditi method, showed no spirochetes.)

Otitic Pathology—Right Ear:

Middle Ear: Edematous submucosa. Meconium present in tympanic cavity.

Inner Ear: Beginning degeneration of cochlear and vestibular ganglion cells. Productive periostitis producing bony irregularity of semicircular canals.

Left Ear: Changes essentially same as on right. Productive periostitis involving vestibular system very definitely present.

CASE 16.—B. T. Autopsy No. 3924. Negro male, still-born.

Family History: Mother gravida II, age 19. No miscarriages. Husband has "bad blood" and refuses treatment.

Present History: Normal pregnancy. Full-term infant, still-born October 12, 1929. Spontaneously delivered, weight six pounds.

Autopsy Diagnosis: (1) Prematurity. (2) Still-born.

Otitic Pathology—Right Ear:

Middle Ear: Mesenchymal remnants present in tympanic cavity. Stapedial foot-plate is bowed inward.

Inner Ear: Productive periostitis present. Enchondral layer of bone in otic capsule is abnormal.

Left Ear: Essentially same changes as in right ear.

DESCRIPTION OF HISTOPATHOLOGICAL CHANGES

As stated above, syphilitic disease of the middle and inner ear is manifested by several definite and quite constant pathologic processes. These may be summarized as follows:

1. *Productive Periostitis (nongummatous).* This was one of the most common findings in the Washington University series. It was present in both the prenatal and acquired cases. It most commonly involved the semicircular canals, with no special predilection for any one canal. Crus commune and ampullated extremities were involved, as well as the limbs and nonampullated ends. Ductus endolymphaticus was markedly involved in one case.

The degree of involvement of the vestibular canal system, however, was quite variable. In some cases, a slight irregularity in contour with roughness and only slight invasion of the perilymphatic lumen was the only manifestation of productive periostitis. In other cases the entire perilymph space was filled with fibrous tissue and the bony lumen was highly irregular. The endolymphatic channel appeared to be very resistant to the invasive process and remained intact and empty in all but two or three areas in the entire series. The most marked invasion occurred in Case 3 (4250) in which the entire semicircular canal has been filled with new-bone formation, in one area. A microfracture has occurred through the otic capsule surrounding this canal.

In the Washington University temporal bone collection there has been found only one case showing a bony cast of the vestibular labyrinth, with no positive syphilitic history (No. 7354). In the older literature this late stage of productive periostitis (bony cast formation) was frequently ascribed to chronic purulent labyrinthitis.

2. *Deformity of the Stapedial Foot-plate:* A bowing inward or outward of the foot-plate of the stapes was evident in most of the cases, both prenatal and acquired. Deformities of the crura were also noticed frequently.

3. *Atrophy of the Spiral Ganglion Cells and Organ of Corti:* Atrophy and degeneration of the organ of Corti was present in variable degree in almost every case. It was usually not completely atrophied in all the turns of the cochlea, however.

Atrophic changes in the spiral ganglion cells, Rosenthal's canal, and Reissner's membrane were seen frequently.

4. *Round Cell Infiltration:* Accumulations of round cells in the cochlea along the basement membrane, in the stria vascularis, in the spiral ligament, in the modiolus, and in the internal auditory meatus along the fibers of the eighth nerve were seen frequently. Some of these masses of round cells were perivascular in arrangement and others were not. Whether such accumulations of lymphocytes may be classified as "miliary gummata" (as they were by Mayer and Fraser) is a debatable question.

5. *Vascular Changes:* In a few instances, among the prenatal group, areas of diapedesis around the veins in the subarcuate fossa were present. Obliterative endarteritis was observed among the acquired cases.

In several of the prenatal cases in which there was an associated luetic hemorrhagic disease of the newborn, marked vascular congestion, and areas of frank hemorrhage into the perilymphatic channels of the vestibular and cochlear branches of the membranous labyrinth were observed.

6. *Changes in Otic Capsule:* True "gummatous" osteitis with definite gummata was not found in any of the cases. However, pathological alteration of staining in the otic capsule without gummata was seen in a number of sections.

DISCUSSION

PATHOLOGIC CHANGES IN THE TEMPORAL BONES IN PRENATAL AND ACQUIRED LUES

Otto Mayer and J. S. Fraser gave an excellent resume of the histopathology of the ear in late congenital lues. They considered the bone lesions to be of primary importance in producing many of the membranous changes. The three distinct bone pathological pictures described were (1) osteomyelitis gummosa; (2) periostitis gummosa, and (3) periostitis productiva (non-gummatous).

In addition to the bone lesions, they stressed the finding of frequent gummata, both the classical variety of the gumma and the so-called "miliary gumma." The latter was the one most commonly found by Mayer and Fraser. Therefore they used the term "gumma" for any area ranging from the classic focus with Langhans giant cells and epithelioid cells surrounding a necrotic center to a simple mass of lymphocytes arranged around a capillary. Mayer and Fraser quoted Freund's work on the pathology of bone syphilis as an authoritative source for such a conception.

Freund's study on bone syphilis showed that in luetic lesions of bone round-cell accumulations must be considered miliary gummata, since pathogenetically they are early forms of more classic forms of the gumma.

Otosclerosis and Syphilis: In 1927, the late Gustav Alexander presented a paper before the Otological Section of the New York Academy of Medicine in which he described the histopathology of the ear in six cases of acquired lues. In this paper he mentioned the occurrence of cochlear end-organ atrophy in lues and ascribed this atrophy to an obliterative endarteritis of the terminal branches of the internal auditory artery, as well as to the effect of bone changes in the otic capsule which affected the neuro-epithelium by pressure. However, it is surprising to note that in one of these six cases, Alexander claimed to have demonstrated "otosclerotic foci" which he describes as "gummatous." As a result of this finding, Alexander concludes "* * * otosclerosis may be caused by lues * * * and this finding gives me a base for saying that (1) in the future I shall use antiluetic treatment with neosalvarsan and hydrargyrum and also the malaria treatment of Wagner in cases of otosclerosis." He goes on to urge further, that children of otosclerotic families should be given prophylactic antiluetic treatment, and that pregnant otosclerotic women be given antiluetic therapy.

On the basis of the findings in the present study, the writer cannot agree with Alexander's dictum stated above. In Case 8 (5204), typical otosclerotic "herde" were demonstrated in the classic site of predilection in both ears, and the patient had syphilis. However, the characteristic productive periostitis of lues had also made its imprint upon the temporal bone picture in this case, with resultant irregularity and bony invasion of the semicircular canals. One sees, therefore, two distinct and separate pathologic lesions in the same patient. A causal relationship between syphilis and otosclerosis is not necessarily present. It is interesting to note that in Case 8 there was no stapedial ankylosis and the audiogram shows an accordingly minimal hearing impairment (Fig. 12). Neither the syphilis nor the otosclerosis has damaged the hearing to any appreciable extent.

In the sixteen case reports of syphilis of the ear described earlier, we find only one in which otosclerosis was present also.

Furthermore, of the four cases of otosclerosis in the temporal bone collection of Dr. Dorothy Wolff, only one (Case 8 described above), had syphilis. In none of the other three cases of oto-

sclerosis was it possible to find bone changes similar to luetic productive periostitis.

Accordingly, it is neither statistically nor pathologically justifiable to attach any causal relationship of syphilis to otosclerosis, in so far as the present study is concerned. Alexander's radical therapeutic suggestions seem to have been made on very feeble pathologic evidence.

Effect on Hearing: A great variety of opinion prevails in relation to the problem of effect of lues upon hearing. In the earlier days of otology, syphilis was considered one of the prime causes of chronic progressive deafness.

In 1911, however, in a serologic study of seventy cases of chronic progressive deafness, Zange concludes that syphilis did not play an important part in the etiology of either labyrinthine (nerve) deafness or otosclerosis.

The more conservative and rational belief of most otologists today is that syphilis is not a remarkably important cause of either nerve or conduction deafness.

C. C. Bunch (1931), observed that the loss in acuity for tones of high pitch with advancing age "is a constant factor in patients in whom a clinical diagnosis of arteriosclerosis, hypertension, chronic cardiac conditions, syphilis or malignant conditions has been made. No one of these more or less generalized systemic conditions can be considered responsible for this loss."

Ciocco (1937), concludes that syphilis apparently does not cause "nerve deafness" more often than does any other disease. Hearing impairment among luetics is more frequent in neurosyphilitics.

Reference has been made above to the conclusions of Crowe, Guild and Polvogt in regard to syphilis and high tone deafness.

In discussing the prognosis for recovery of hearing in cases of luetic nerve deafness, Moore states that only in hearing impairment associated with early meningeal neurosyphilis may one expect any therapeutic results. Such cases are characterized by a sudden onset of hearing loss, with no involvement of the vestibular apparatus, and with serologic evidence of acute syphilitic meningeal irritation. Intensive arsphenamine therapy in such cases may give a return of hearing to normal levels in more than 50 per cent of cases. However, in cases of late congenital or acquired neurosyphilis, such

therapy is almost hopeless. Doubtful results have been reported in the latter group following fever therapy.

Moore's analysis of results of therapy correspond closely to what one would expect in view of the pathological process in the temporal bone, since in the latter group of cases (late congenital and acquired) there is an irreversible osteitis and periostitis combined with a degenerative atrophy of neural elements both in the spiral ganglion and in the cochlear end-organ.

Reliable audiograms were obtained in three of the acquired cases (adults) in the Washington University series. They are represented in Figs. 1, 11 and 12. The audiograms were not obtained in a sound-proof room, but the patients were intelligent and co-operated well during the tests. Taking into consideration the masking effect of ward noises, there seems to be no very definite type of hearing loss in either patient which would bear the description of either "nerve" or "conduction" type of deafness. The findings correspond to what Bunch and Ciocco both observed, namely, that there is no specific type of hearing loss associated with syphilis, in middle-aged patients, especially.

In Case 1 (age 49) Fig. 1, there is a tone gap for the right ear at 4096 D. V. Such a gap corresponds closely with the type described by Bunch as due to acoustic trauma.

In Case 6 (age 63) Fig. 11, all tones are heard with the exception of 8192 D. V. on the right. The threshold throughout the range is somewhat lowered and bone conduction is somewhat decreased. However, such a hearing loss is not surprising in a 63-year-old man afflicted with an esophageal neoplasm, and his hearing loss cannot be justifiably accredited to his lues alone.

In Case 8 (age 45) Fig. 12 (otosclerosis and syphilis), all tones are heard by both ears. The somewhat lowered threshold is not very significant in view of the masking effect of ordinary ward noises.

SUMMARY AND CONCLUSIONS

The temporal bones of sixteen cases of syphilis were studied histologically. Eight cases were acquired (adults) and eight prenatal (infants).

The following distinct histopathologic changes were found quite constantly.

1. Productive periostitis (luetic) producing invasive fibrosis followed by ossification of the perilymphatic and endolymphatic channels of the vestibular apparatus. Malformations of the bony canals are also produced.

2. Bony abnormalities of the stapes, especially of the foot-plate.

3. Atrophy of the neuroepithelial elements of the cochlear and vestibular systems.

4. Lymphocytic (small round cell) infiltration of the cochlea and spiral ganglion (miliary gummata).

5. Vascular changes, consiting of diapedesis in prenatal cases, and obliterative endarteritis in acquired cases.

There is apparently no specific variety of hearing loss associated with luetic aural disease.

Evidence has been presented to show the lack of causal relationship between syphilis and otosclerosis.

Grateful acknowledgment is made to Dorothy Wolff, Ph.D., in whose laboratory the research was carried out, and to Dr. L. W. Dean, Head of the Department of Otolaryngology.

BIBLIOGRAPHY

1. Alexander, Gustav: New Histopathological Findings in the Ear in Lues and Their Importance in General Pathology of the Ear. Laryngoscope, 38:295-305 (May), 1928.

2. Alexander, Gustav: Zur Frage der Luetischen Erkrankungen des Labyrinthes und des Hörnerven. Wien. Klin. Wochenschrift, 24:13, 1911.

3. Alexander, Gustav: Die Ohren-Krankheiten im Kindesalter. F. C. W. Vogel, Leipzig, 1912.

4. Andersen, H. C.: Ein Fall von kongenitalem syphilitischem Ohrenleiden mit klinischer und pathologischer Untersuchung. Acta Otolaryngologica, 25:37-50, 1937.

5. Baratoux: Quoted by Schwartze. (See below.)

6. Beck, O.: Die Krankheiten des Gehörorgans. A. Denker and O. Kahler. Julius Springer, Berlin, 1926.

7. Brühl, Gustav: Beitrage zur Pathologischen Anatomie des Gehörorgans. Zeitschrift fur Ohrenheilkunde, 52:232-236, 1906.

8. Bunch, C. C.: Further Observations on Age Variations in Auditory Acuity. Arch. Otolaryng., 13:170-180 (Feb.), 1931.

9. Christie, J. F.: Syphilitic Infection of the Ear. Med. Press & Circular, 5:189 (Oct.), 1938.

10. Ciocco, Antonio: Nerve Deafness from Syphilis. Laryngoscope, 47:572-576, 1937.

11. Cooke, J. V., and Jeans, P. C.: Prebuscent Syphilis. D. Appleton & Co., New York, 1930.

12. Crowe, S. J., Guild, S. R., and Polvogt, L. M.: Observations on the Pathology of High Tone Deafness. Bull. Johns Hopkins Hospital, 54:315-381 (May), 1934.

13. Downie, Walker: Ein Fall von erworbener totaler Taubheit in Folge von hereditarer syphilis, mit Sectionsbericht. Zeitschrift für Ohrenheilkunde, 30:236-241, 1897.

14. Fraser, J. S., and Muir, Richard: The Pathology of Congenital Syphilitic Disease of the Ear. J. Laryng., Rhino. & Oto., 32:8-30 (Jan.), 1917.

15. Freund, Ernst: Über Knochensyphilis. Virchow Archiv für Pathologische Anatomie und Physiologie, 288:146-211 (Feb.), 1933.

16. Grünberg, Karl: Uber Spirochatenbefunde im Felsenbein eines Luetischen Fötus. Zeitschrift für Ohrenheilkunde, 63:223-228, 1911.

17. Mayer, Otto: Zur Kenntnis der Pathologischen Veränderungen im Gehörorgan bei der Lues Congenita Tarda. Zeitschrift für Hals-nasen-und Ohrenheilkunde, 37:2-30, 1935.

18. Mayer, Otto, and Fraser, J. S.: Congenital Syphilis. J. Laryng. & Otol., 51:683-714; 755-778 (Nov.), 1936.

19. Moore, J. E.: The Modern Treatment of Syphilis. Charles C. Thomas, 1933.

20. Ruskin, S. L., and Hyslop, G. H.: Acute Syphilis of Inner Ear. Laryngoscope, 48:280-285 (April), 1938.

21. Schlittler, E.: Klinische und Histologische Untersuchungen bei Kongenitaler Labyrinther Syphilis, ein Beitrag zur Pathogenese der Kogenital-luetischen Innenohrerkrankung. Zeitschrift für Hals-Nasen-und Ohrenheilkunde, 21:490-515 (May), 1928.

22. Schlittler, E.: Die Syphilitischen Erkrankungen des Innenohres (ubersichtsreferat). Zentralblatt fur Hals-Nasen-und Ohrenheilkunde, 27:337-349, 1936.

23. Schwartze, Hermann: Handbuch der Ohrenheilkunde. F. C. W. Vogel, Leipzig, 1892.

24. Stokes, J. H.: Modern Clinical Syphilology. W. H. Saunders Co., 1935.

25. Wittmaack, K.: Gehörorgan, Band XII. Handbuch der Speziellen Pathologischen Anatomie und Histologie, Henke and Lubarsch. Julius Springer, Berlin, 1926.

26. Zange, J.: Chronische Progressive Schwerhörigkeit und Wassermannsche Seroreaktion. Zeitschrift für Ohrenheilkunde, 62:1-18, 1911.

LIX

SOME PHASES OF TUBERCULOSIS OF THE LARYNX*

MERVIN C. MYERSON, M.D.

NEW YORK

Our present-day understanding of tuberculosis of the larynx is due to the progress which has been made as a result of the invention of the laryngeal mirror and the roentgen-ray, and the discovery of the tubercle bacillus.

Tuberculosis of the larynx is always secondary to disease of the lung. When the existence of a primary tuberculosis of the larynx is claimed, it would be well to take into consideration the fact that our present-day methods for the detection of pulmonary tuberculosis are inadequate in some cases to enable us to establish the lung as the seat of the primary focus.

The method by which the larynx is involved has always been a subject of controversy. I am strongly inclined to the opinion that the larynx is invaded by way of the blood stream or the lymphatics. A lymphatic pathway between the lung and larynx has not yet been demonstrated. As yet no proof has been offered against the hematogenous pathway. It is conceivable that transient bacteremia occurs in the acutely ill tuberculous, for it is under such circumstances that organs other than the larynx are frequently involved.

In favor of the hematogenous spread are the cases of lingual tuberculosis in which the location of the lesion is such that sputum contact does not occur. The fact that there are more larynges bathed in bacillus laden sputum which continue uninvolved than those which become infected is a strong argument against the contact theory. The very evident hematogenous type of infection is seen in the diffusely hyperemic and slightly thickened larynx, which is caused by a miliary spread.

A series of one thousand successive cases were studied and analyzed. These cases were seen during the period of five and a half

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years between January, 1933, and July, 1938. Only ambulant patients were included in this study. The proportion of males to females was 1.33 to one. This agrees with the experience of others. Our youngest patient was a boy 10 years of age, while our oldest was a man of 76 years.

I have arranged tuberculous lesions of the larynx into three groups: The exudative, the productive and the destructive. The following classification of pathologic changes is suggested:

A. Exudative } Infiltrative
B. Productive }

a. Cellular

1. Diffuse or nodular
2. Miliary

b. Fluid (edema)

1. Proliferative

Hyperplasia (pachydermia)
Papilloma
Polyp
Tuberculoma
Perichondritis and chondritis
Fibrosis

C. Destructive

1. Ulceration
2. Necrosis

Our studies indicate that the posterior structures of the larynx are most frequently involved. This agrees with the findings of others. The following is an analysis of the lesions encountered in this study. The tabulations represent the lesions and their distribution, which were found in the larynx at the first examination. When similar bilateral structures were involved, only one was counted.

Very few larynges have the same lesions in the same locations. The ten locations in which these lesions may be found are listed in the following table. The tissue changes are represented by cellular and edematous infiltration, ulceration, tuberculoma, epithelial hyperplasia (pachydermia), papilloma, polyp, perichondritis, chondritis,

fibrosis and necrosis. These various manifestations are more likely to occur in certain parts of the larynx than in others. Moreover, the lesions are likely to be extensive and diffuse rather than single and isolated.

TABLE I

DISTRIBUTION OF LARYNGEAL LESIONS
(1,000 cases)

	Infiltration	Edema	Ulceration	Tuberculoma	Total
*Arytenoids	202	235	82	5	524
Interarytenoid space	155	28	66	110	359
*Vocal cords	223	17	90	11	341
**Epiglottis	134	82	106	2	324
*Ventricular bands	161	58	79	8	306
Aryepiglottic folds	36	49	7	0	92
Vocal processes	7	0	55	2	64
Ventricle	0	7	0	9	16
Anterior commissure	0	0	1	1	2
Postcricoid	0	0	2	0	2
Subglottic	0	0	1	0	1
Epiglottis-papilloma					1
Interarytenoid papilloma					1
Abscess of arytenoid					1
Fixation of cord: Right—7; Left—13; Both—4.					
Diffuse hyperemia					25
					2,039

*One or both structures. **Ulceration includes necrosis.

TABLE II

ANALYSIS OF SOLITARY OR ISOLATED LESIONS

	Infiltration	Ulceration	Edema	Tuberculoma	Total
Interarytenoid space	18	2	4	26	50
Epiglottis	6	17	5	1	29
*Arytenoid	3	5	1	1	10
*Vocal process	1	7	0	0	8
*Vocal cord	5	4	0	1	10
Ventricular band	4	1	1	3	9
					116

*One or both.

TABLE III

OTHER OTOLARYNGOLOGICAL TUBERCULOUS LESIONS IN THIS SERIES

Pharynx	25
Otitis media	16
Tongue	13
Mastoiditis	9
Tonsils	7
Nose	5
Oral cavity	5
Palate	2
Lip	1
Zygoma	1

GENERAL PATHOLOGICAL CONSIDERATIONS

The larynx is secondarily involved in an individual who has been sensitized to the tubercle bacillus at the time of the primary infection. Due to a reactivation of a focus in the lung, the bacilli find their way into the larynx.

Most authors agree with Ranke,[16] Krause,[10] Rickmann,[19] and Safranek,[20] that localization in the larynx and the consequent tissue reaction constitutes an allergic phenomenon. This allergy indicates a defensive mechanism which is represented in the response of the sensitized tissues to the presence of the antigen, which is the tubercle bacillus. The inflammatory reaction inhibits the progress of the effects of the invasion of the bacilli. In addition to the hypersensitiveness or allergic state, the larynx is influenced in its response by such factors as the age and general condition of the patient.

The involvement is very likely produced by a generalization of tubercle bacilli in the blood stream, with localization in the larynx. This usually occurs from an extra pulmonary focus, such as a kidney or bone lesion, or from a cavity in the lung from which the tubercle bacilli reach the blood stream by way of the lymphatics.

The cellular infiltration which occurs in the subepithelial tissues may be localized to a single structure, such as a vocal cord or ventricular band, or it may be widespread and involve most or all of the laryngeal structures. If one examines the larynx very early in its involvement he will frequently see a diffuse and intense hyperemia, which may be localized to one or all of the structures. Although there is intense redness, there is no pain. In many instances the hyperemia disappears and the larynx does not become involved

in what is recognized as a tuberculous process. This hyperemia is evidence of the early exudative phase and is the earliest evidence of tuberculosis of the larynx. If one watches such a larynx daily he will either see a disappearance of the hyperemia and a resolution of the pathological process, or he will detect a beginning elevation and gradual destruction of a localized area of the epithelium which results in an ulcer. The first is more frequently the case. At times the progress of the early lesion is so rapid that the hyperemia is overlooked and the first sign of involvement is the ulceration. If the ulceration is seen early, however, there is usually some remaining hyperemia in the vicinity. In the region of the vocal processes and on the vocal cords, ulcers frequently appear, with little evidence of thickening to denote the antecedent subepithelial infiltration.

The edematous infiltration is not preceded by a hyperemic change and is slower in its progress. One can observe the tissues involved in the edematous infiltration undergoing a gradual thinning process which represents absorption and resolution, followed by subepithelial fibrotic changes.

Because it is composed of cartilages which are covered by soft tissues, some of which are closely related to the supporting structures, while others are separated from them by considerable loose areolar tissue, the reaction of the laryngeal tissues to injury by the tubercle bacillus is quite different than that of the lung.

While it is true that the nodular tubercle is the typical tuberculous lesion, it is interesting to note that it is seldom found in the larynx. In the larynx, as in the lung, the tubercle bacillus calls forth a specific type of reaction. This is an exudative reaction which may later become proliferative or destructive. The local reaction in the larynx may represent the tissue response to a fresh localization, or may be due to extension from a pre-existing, contiguous lesion.

There is a type of nodular response which is analogous to the primary response in which we find well formed tubercles. Analysis of infections of this type indicates that the focus in the lung is usually more or less inactive. Very frequently the patient does not know that he has a pulmonary condition until he is made aware of it by the involvement of his larynx. In this group belong the localized infiltrations of the vocal cord, the epiglottis, the ventricle and the interarytenoid space, which are slow in their evolution and remain inactive and more or less stationary until something occurs to extend the lesion or create new lesions in other parts of the larynx.

The exudative reaction in tuberculosis of the larynx is no different than such reaction elsewhere. At the beginning of the inflammation the blood stream sends forth its elements which find their way into the interstitial tissues. The tissues are filled with fluid (serum) and a large number of leucocytes. The fluid may be predominantly fibrin forming. Cellular elements are introduced into the exudate which are to designate the future tuberculous process. If the exudation is of small quantity and extent, resolution and a return to normal will occur. With the introduction of the small round and the epithelioid cells, together with an occasional giant cell, the exudate tends to organize. Organization brings the productive or proliferative stage. This stage represents the process of infiltration and its sequelæ.

If, however, organization does not occur, or the process of organization is weak, the productive stage does not assert itself, and instead of organization and a tendency toward healing, the destructive stage supervenes. This is characterized by eventual ulceration or necrosis.

The exudative process may be predominantly cellular or fluid. When the reaction is severe, the lesion is diffuse and widespread, so that several structures, such as the epiglottis, aryepiglottic folds and arytenoid areas, may be attacked simultaneously. At times additional structures are involved. When the antigen or offending substance is small in quantity, the reaction is more localized and more benign; in fact, it may be so small as to pass unnoticed.

This early cellular reaction frequently occurs as an extensive process in postmortem studies. At times these changes proceed rapidly to the destructive stage, so that well defined ulceration is present. These changes do not represent lesions of the larynx which existed during life. They are, rather, the result of changes which occurred just before death when a spread occurred to the laryngeal and other structures by way of the blood stream. We have examined larynges which were free from evidence of disease a day or two before death, yet at autopsy they presented extensive ulcerations.

The lesions may be classified according to the severity of the reaction, as: severe, acute and rapid; intermediate or subacute; slow, or chronic. The first very frequently results in ulceration and necrosis; the last two are likely to end in fibrosis and healing. Organization of exudate indicates small dosage of the infecting organism, good resistance, and eventual fibrosis, unless further generalization and localization take place, or the local lesion is activated. The

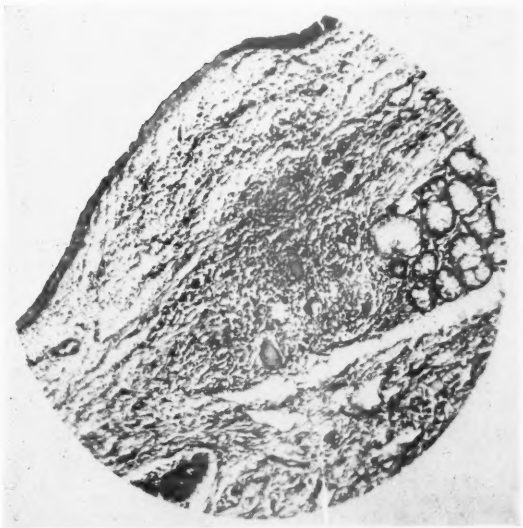


Fig. 1. Section of epiglottis demonstrating subepithelial tubercle formation in center of field. Several Langhan's giant cells are present.

proliferative changes are represented by a variety of hyperplastic changes in the mucous membrane. Proliferative changes in the connective tissue areas occur by virtue of the conversion of the epithelioid cells into connective tissue cells and eventual firm, fibrous tissue.

Varying degrees of infiltration may exist in the same structure. In a single low power field one can frequently see healing and repair alongside of destructive changes. Destructive changes extend deeply by virtue of degenerative changes which attack the tuberculous infiltration. Caseation occurs and the microscopic sections show cellular and nuclear remnants interspersed among the collagen and hyaline fibers which originated from the epithelioid cells.

HISTOPATHOLOGICAL CONSIDERATIONS

Cellular Infiltration: The epithelium is frequently greatly thickened by the stimulation of the subepithelial cellular infiltration and its by-products. Papillary indentations are seen in the region of the

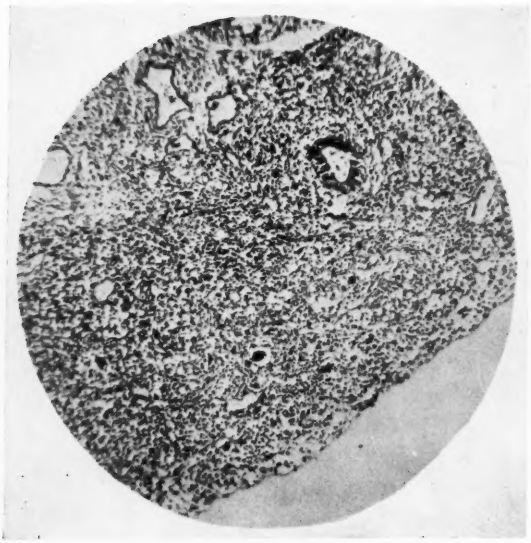


Fig. 2. Photomicrograph of section through the ventricular band. There are numerous dilated and distorted mucous glands scattered throughout the tuberculous process.

basement membrane of the squamous epithelium, causing the base of the epithelial layer to appear in undulations. The infiltration is most frequently diffuse. Here and there it may become localized. Giant cells are more frequently absent than present.

The blood vessels are usually surrounded by groups of cells, among which the small lymphocytes predominate. The intima of the arteries is unaffected. In long standing and extensive lesions the smaller arteries are indented and even obliterated by the lymphocytic infiltration. The adventitia of the larger arteries is greatly thickened because of an increase of its fibrous tissue elements. The veins are occasionally seen filled with thrombi in which small lymphocytes predominate. The small capillaries are especially liable to such thrombus formation. It is easy to mistake these smaller capillaries for lymphatic channels. We have not encountered lymphatic vessels involved by such thrombosis.

The periglandular spaces are markedly involved by the cellular infiltration. The epithelial elements of the glands may degenerate

because of invasion from without, but are never attacked from within. Gland ducts are seen to be obliterated and distorted, and occasionally one becomes cystic because of superficial duct occlusion. At times the infiltration forces its way between the individual glands so as to separate them. The interference with glandular function by the tuberculous process accounts for the very frequent complaint of dryness among the patients afflicted with laryngeal tuberculosis.

There is cellular invasion around the muscle bundles and occasionally even around the individual fibers, so that the muscle bundles and fibers may become dispersed and separated. The infiltration is purely interstitial, for it is never seen in the muscle fibers themselves. Atrophy of the muscles is at times seen in association with these changes, denoting a long standing process.

The perineural spaces are affected by the infiltration. At times the sheath is involved and partly destroyed by the infiltrative process. The nerves are destroyed only when a destructive process rapidly passes through the entire involved structure. Some have claimed that the infiltration of the neural sheath accounts for painful swallowing even in the absence of ulceration.

Fluid Infiltration or Edema: This form of infiltration is frequently seen in the loose cellular structures of the larynx. It is a more typically allergic reaction than the cellular infiltration. The edema usually appears as a transparent, and at times finely granular process. It invades both the extracellular and intracellular spaces. It is more prominent and more readily recognized in the interstitial connective tissue areas, where it so separates the fibers as to render the connective tissue unrecognizable. The connective tissue cells are pale and swollen, as a consequence of which their size is greatly increased. One recognizes a distention and swelling in the individual cell, as a result of which the cell protoplasm loses its stain or stains poorly. The cells may have no stain whatsoever, or be replaced by collagen or hyaline masses. Most of these infiltrations have few elements to indicate the presence of tuberculosis. There are, however, in most of these processes, cellular elements which indicate the true nature of the lesion. When cellular elements are present in large quantities, ulceration is liable to occur. In the later stages of the disease the spaces between the connective tissue fibers may contain collagen and hyaline masses.

There are also present fine walled endothelial vessels which are filled with finely granular or homogeneous material. These appear to be small capillaries rather than lymph vessels. Most of them

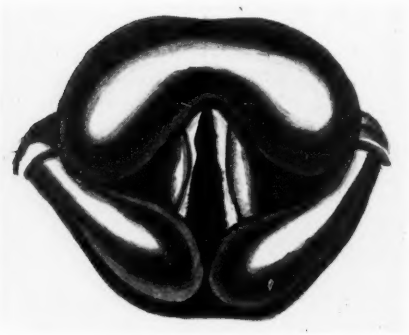


Fig. 3. Semi-diagrammatic drawing of a larynx involved in a widespread and intense edematous infiltration.

contain red blood cells or degenerated red blood cells or serum, not tuberculous products.

Fibrin Formation: This is found especially in the edematous connective tissue areas. It is characterized by small or large collections of fibers, with small granules interspersed among them. The fibrin fibers are usually parallel in arrangement, but frequently cross each other. They are easily recognized, definitely wavy and coarse, and should not be confused with collagen or hyaline masses, or fibrous connective tissue. They are always associated with edematous areas and with cellular collections. The presence of fibrin indicates a relatively early or recent lesion. The edema and fibrin occupy areas which are free from cellular infiltration or tubercles, and should be considered a by-product of the active cellular process close by.

Ulceration: This is a fairly frequent result of the subepithelial tuberculous infiltration. The subepithelial process causes degeneration of the superficial layers of the mucous membrane which are gradually shed to form an ulcer. The first response consists of a thickening of the epithelial layer, which later is followed by this degenerative process. It is this mechanism rather than a true pressure effect which causes the ulcer. The squamous epithelium offers much more resistance than the columnar to this degenerative process. This is recognized in the varying degrees of hyperplasia which

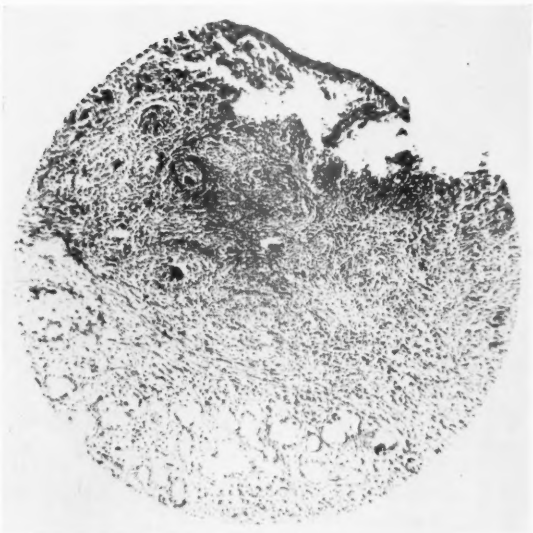


Fig. 4. Photomicrograph of section through the ventricular band. The field shows ulceration, overhanging epithelium, extensive subepithelial tuberculous infiltration and scattered giant cells.

occur in the squamous epithelium and which many writers have called pachydermia.

The superficial epithelial cells can be seen disintegrating in many of the sections. They have lost their nuclei, are poorly stained, and are being shed. When they are completely lost the small lymphocytes and epithelioid cells make their appearance in the denuded area. As the cellular infiltration becomes exposed, there may be a concomitant necrotic change in the deeper structures. The deeper tuberculous granulation tissue keeps pace with the size and depth of the ulceration. The larger ulcers have an exudative surface covering which is granular and without form. This is of the same structure and composition as the superficial, exudative, necrotic layer which is seen in extensive ulcerogranulomatous lesions of the bronchus with caseation.

Because the ulcer may extend downward to or even beyond the perichondrium, the laryngologist is not always able to ascertain

TABLE IV

ULCERATIONS ACCORDING TO STRUCTURES

Epiglottis	116
Arytenoids	82
Vocal cords	90
Ventricular bands	79
Interarytenoid space	66
Vocal processes	55
Aryepiglottic folds	9
Postcricoid area	2
Anterior commissure	1
Subglottic area	1
Total	501

ULCERATIONS AS ISOLATED LESIONS

Epiglottis	17
Vocal processes	7
Arytenoids	5
Vocal cords	4
Ventricular bands	1
Interarytenoid space	2
Total	36

the depth of such a process by mirror examination. He must therefore be guided by his understanding of the pathological process.

Microscopically the superficial ulcer shows no sharp line of demarcation from the surrounding healthy epithelium. The small lymphocytes and epithelioid cells gradually thin out as the surrounding zone of healthy tissue is reached. If the process is not progressive, it is quickly limited by the granulation tissue, which eventually gives way to fibrous tissue. If it continues, however, a deep ulcer, with well defined edges, results. Extension of this ulcerative process can involve the perichondrium and cartilage with a resulting necrosis.

The deep ulcer may be indolent or active. Most frequently it is active. We regard this type of active ulcer as a disintegrating lesion. It denotes a lack of local and general resistance. It is progressive and bespeaks an unfavorable prognosis. These disintegrating lesions have a fine, granular base which is indicative of tuberculous cellular deposits. When this type of lesion is generalized throughout the larynx, the infection is regarded as of the miliary type.

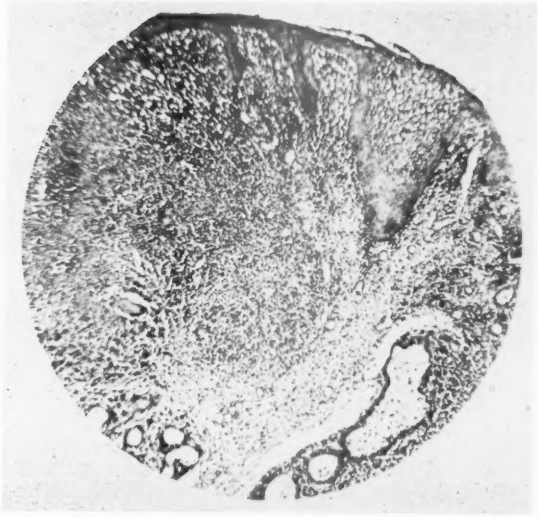


Fig. 5. Photomicrograph of section of a vocal cord, showing pachydermic hyperplasia with extensive cellular infiltration beneath the epithelium.

Pachydermia: This term, as it is applied to the stratified, squamous epithelial changes in tuberculosis of the larynx, does not indicate the pachydermia which is seen in chronic irritations of mucous membranes. It is a marked hyperplastic process. Pachydermia does not occur in columnar epithelium; it does, however, occur after this type of epithelium has been transformed into the cuboidal or squamous type. The most frequent locations of this change are the vocal cords, the interarytenoid space and the ventricular bands. The epithelium and the subjacent connective tissue is arranged in the form of papillæ. At times, epithelial metasplasia is seen. The epithelial cells extend deeply into the connective tissue in the form of pegs and are surrounded by the tuberculous elements. These pegs indicate an effort on the part of the epithelium to keep the tuberculous process at a distance. If the process is sufficiently active the epithelium is invaded and ulceration occurs, at times leaving islands of hyperplastic epithelium in the ulcerated area.

This hyperplastic area is a phenomenon of the superficial epithelial layers, denoting a special type of reaction to the tuberculous changes nearby. The squamous epithelial cells are firmly bound by their intercellular fibrils. In the subjacent structure tubercles or

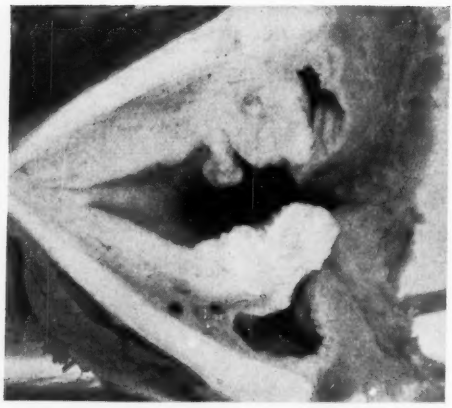


Fig. 6. Coronal section through the level of the ventricular bands of a male adult larynx viewed from above. There is marked thickening of the soft tissues, especially in the region of the arytenoids, and the infiltration extends to the inner perichondrium of the thyroid cartilage. There is a granulomatous polyp springing from the region of the right vocal process.

diffuse cellular infiltrations are encountered. At times giant cells are seen in close proximity to the hyperplastic mucous membrane. The subjacent structure shows a tendency to fibrosis which can eventually force the epithelial cells apart and so disturb them as to cause their destruction.

Papilloma: Small papillary elevations are frequently seen in tuberculous disease of the larynx. As a result of an increase of the epithelial elements, a true papillomatous proliferation may occur. In such cases the examiner sees the regular groups of projecting masses of epithelium superimposed upon a subepithelial structure which is markedly infiltrated with tuberculous cellular elements.

Polyp or Polypoid Reaction: Polypoid changes and formation of polyps occasionally occur with the edematous infiltration of the larynx of younger individuals. The polyp is in reality a neoplasm of tuberculous nature which is usually not recognized as such. These tumors are round, circumscribed masses which may be solitary, or occur in groups. Their size varies from one to ten millimeters in diameter. They are covered with mucous membrane and may be long and slender, or short and broad. Their color is pale red, pink or grayish-white. They are readily movable and may be

TABLE V

DISTRIBUTION OF TUBERCULOMATA

Interarytenoid space	110
Vocal cords	11
Ventricular bands	8
Ventricles	9
Arytenoids	5
Epiglottis	2
Vocal processes	3
Anterior commissure	1
Total	149

TUBERCULOMATA AS SOLITARY LESIONS

Interarytenoid space	26
Ventricular bands	3
Vocal cords	1
Arytenoids	1
Epiglottis	1
Total	32

expelled by strong coughing. These tumors are usually considered benign polyps at the time of their removal. Both the laryngologist and patient are surprised when the laboratory reports their tuberculous nature. They usually occur on the vocal cord, but may originate from the area of the cushion of the epiglottis, in which case they may cover or fill the anterior commissure. They also occur in the ventricle and interarytenoid space.

The production of these polyps can be traced to a localized hyperplasia of the mucous membrane which lifts the nutrient submucous tissue with it as it is projected away from the main structure. The pedicle may be soft or firm; this is also true of the tumor. Andral,[1] Mackenzie,[13] Schnitzler[21] and others have reported such polyps. The stroma of these polyps is occupied by the cellular infiltration which is characteristic of tuberculosis.

Tuberculoma: From a purely pathologic point of view, all tumors and tumefactions of a tuberculous nature could be called tuberculomata. Clinically, however, we distinguish it as a granulomatous type of growth with irregular surface and contour, of very small to large size. These growths are usually pale red or dull pink in color. They do not bleed when touched and usually replace or accompany an ulcerous lesion. A tuberculoma is essentially nature's attempt to localize the disease process.

Tuberculomata were found 149 times in this series. They accounted for 7.3 per cent of the collected lesions.

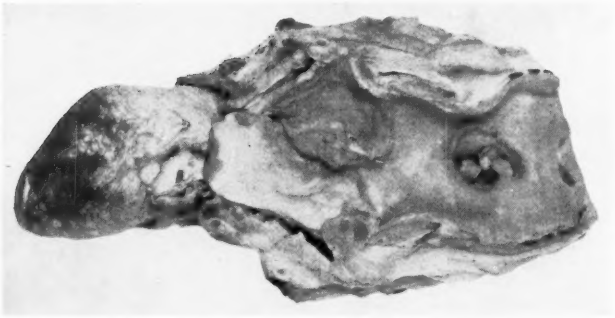


Fig. 7. Autopsy specimen; larynx of a 41-year-old male. There is a large tuberculoma filling the right side of the larynx. Both cricoarytenoid joints were fixed by the infiltrative process in the postcricoid area. A tracheal opening for the tracheotomy tube is present. There is a thickening of the lateral aspect of each side of the larynx, with encroachment upon the pyriform fossæ. A lesion of the left tongue base can be seen.

Tuberculous tumors of the larynx were first described by Tobold[24] in 1866. The older writers recognized these growths in two forms: The granulotuberculoma and the fibrotuberculoma. At the present time there does not appear to be any justification for such classification. The fibrotuberculoma is simply a later stage of the granulotuberculoma. A tuberculoma is always first a granulotuberculoma; as it heals it becomes a fibrotuberculoma because of the increase of its fibrous tissue elements.

These masses extend, remain stationary or disappear, depending upon the local and general resistance. They represent a diffuse cellular infiltration which has broken through the surface and extends above it. In such lesions there are an abundance of small lymphocytes and epithelioid cells, with an occasional giant cell and some fibroblasts. If the tuberculoma shows a tendency to heal, the epithelioid cells and fibroblasts will predominate. As a result, the tissue mass becomes firmer. Necrosis, in the form of caseation, is seen only in superficial layers. The tuberculoma does not always contain tubercles. When tubercles exist they are to be found in the deeper areas. Caseation is more frequently absent than present. There is usually a tendency toward fibrous change in the presence of good resistance, so that there exists a bed of fibrous tissue with granulations upon it. When healing is complete the granuloma is replaced by scar tissue.

Occasionally a tuberculoma may be so large as to obstruct the airway and necessitate the performance of a tracheotomy.

Perichondritis and Chondritis: The first mention of disease of the cartilage and its nourishing membrane, which might have been tuberculous, was traced to Jonstonius (1652) by Monlaur.[14] Petit[15] and Lieutaud[12] reported the results of postmortem studies of advanced tuberculous laryngitis, in which they found ulceration of the larynx and caries of the cartilage. Tuberculosis is the most frequent cause of perichondritis in the larynx. Every type of infiltration is likely to bring with it some perichondrial involvement. Among forty-five cases of perichondritis of the laryngeal cartilages from all causes, Mackenzie found twenty-three were tuberculous.

The perichondrium of any of the cartilages may be involved. Those most frequently involved, in our experience, have been the arytenoids and the epiglottis. The thyroid cartilage is occasionally affected. The cricoid is least frequently involved. Out of 982 infiltrative lesions, the arytenoids accounted for 202, while among 427 edematous lesions the arytenoids were involved 235 times. Perichondritis is a frequent concomitant of these infiltrations. This is indicated by the fact that the structure is thickened in a majoriy of instances after healing takes place. It frequently is deformed. This is readily seen in the epiglottis and arytenoids.

In most instances the perichondrium is attacked by the tuberculous process which is continuous with an ulcer directly above it. Perchondritis, however, frequently exists without ulceration. The cellular infiltration which caused the ulcer may invade the deeper structures and destroy the perichondrium. The perichondrial fibers are then separated by cellular elements, principally small lymphocytes, which fill most of the space between the cartilage and the mucous membrane. This process may further advance to attack the cartilage. One frequently sees a greatly infiltrated and intact perichondrium, which is evidence of its resistance to the local process. Poorly staining cartilage cells and abundant nuclei are seen in the affected areas.

Involvement of the cartilages by an ulcerous necrotic process is relatively infrequent. In this series there were four abscesses of a laryngeal cartilage—in the cricoid, in the thyroid, and twice in the arytenoid cartilage. The course of the production of an abscess of this kind is somewhat as follows: The perichondrium is lifted from the cartilage by purulent exudate which deprives the cartilage of its nourishment. The tissues above are usually edematous, the mucosa

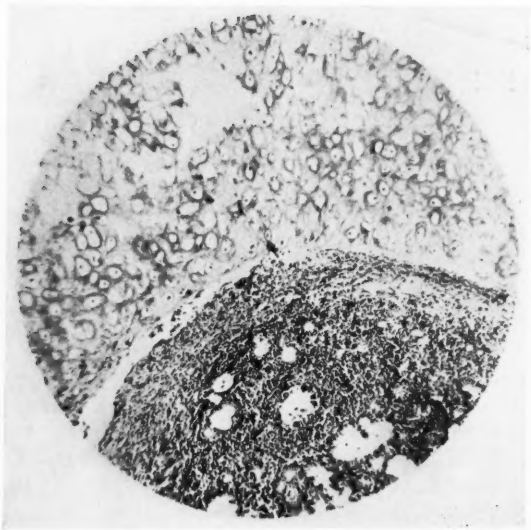


Fig. 8. Photomicrograph of section through epiglottis demonstrating tuberculous invasion of the perichondrium and cartilage.

being tense. The soft tissues are readily lifted from their subjacent, firm structure. The ulcer leading into the necrosed area consists of granulation tissue and is covered with pus. Fistulous tracts may occur. The cartilage now shows ossification and necrosis in the same low power field. As the disintegration of the cartilage progresses, there is considerable softening and loss of identity of the structure. The entire arytenoid cartilage may sequestrate and be extruded. There is usually an increased mobility of the cricoarytenoid joint when necrosis of the arytenoid exists. The muscles may be lifted from their attachments, so that their action is greatly impaired.

Perforation of the laryngeal cartilages by a tuberculous process has been reported by Fletcher,[5] Tyson[25] and others. Tyson reported a case of perforation of the thyroid cartilage and was able to collect seven well defined cases from the literature. The thyroid cartilage

was most frequently affected. One of the cases had a perforation through the thyrohyoid membrane;[17] another expelled an arytenoid cartilage,[1] while in a third there occurred a perforation of the posterior wall of the cricoid cartilage.[18]

Subcutaneous emphyzema is an important symptom of these perforations. The anterior commissure area appears to be the most frequent site of a perforation.

Fibrosis: Fibrosis is the end-result of all lesions which have the capacity to heal. The earliest indication of future healing is the relative increase in the number of epithelioid cells. Where the lesions are characterized by tubercle formation, the healing process is evidenced by a gradual flattening of the peripherally placed epithelioids, and their replacement by connective tissue fibrils. In both the diffuse cellular and nodular forms of infiltration, the epithelioid cells are more abundant, and their conversion into fibrous tissue on a large scale is noted. The tubercles display caseation in their centers. In the tissues which have been affected by edematous infiltration, the connective tissue fibrils appear widely separated. When the fluid is absorbed, fibrosis occurs.

Fibrous tissue formation is seen between the glandular acini. Fibrous tissue also replaces groups of glands or individual glands which may have been destroyed. One encounters atrophy of the glandular capsule in many sections. During the healing process one frequently sees the scattered, small lymphocytic infiltrations with epithelioid cells and occasional giant cells which indicate a newly formed lesion, or a relapse. In some sections tubercles are seen which do not appear to be influenced by the process of healing.

The smaller blood vessels are compressed and frequently obliterated, while the larger vessels present varying degrees of proliferation of their adventitia. In some instances the vessel wall is thicker than the size of its lumen. The compression and obliteration of the smaller subepithelial vessels is thought by some to favor the production of ulcers in the regions supplied by them. I do not believe this to be so, because the occlusion of the vessels is a slow process which permits the circulation of the affected area to adjust itself so as to adequately supply the mucosa.

Ulcerations show evidence of fibrosis in their depths and at their borders. When the ulcer is small and has not extended beyond the basement membrane, there will be no fibrous tissue in the healing, and the defect will be replaced by newly formed squamous epithe-

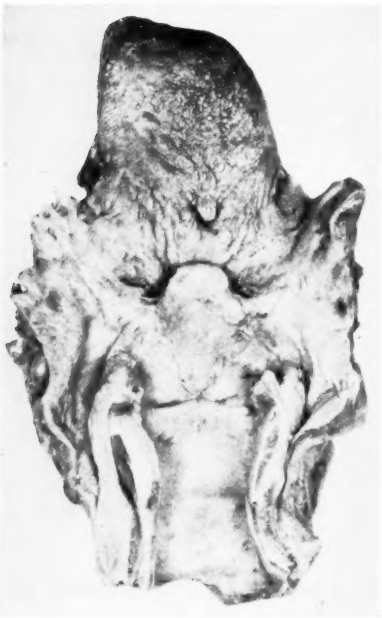


Fig. 9. Autopsy specimen of the larynx of a 25-year-old man. There is extensive destruction of the right side of the larynx which involves the lateral wall of the pyriform sinus. All other structures besides the vocal cords are involved in a diffuse, miliary process.

lium. In the deeper ulcers, however, the entire area is replaced by fibrous tissue.

One is impressed with the solidity and prominence of the fibrous tissue in many fields. This connective tissue formation is not adulterated with tubercles or an infiltrative process. It represents the end-result of the healing.

Miliary Tuberculosis of the Larynx: Acute generalized miliary tuberculosis of the larynx was first mentioned by Isambert.[9] It represents a massive invasion of the larynx by way of the blood stream. When this occurs, not only the larynx, but every organ in the body may be involved. I have seen a generalized spread

Fig. 10. Autopsy specimen of the larynx of a 22-year-old female. The entire interior of the larynx is involved in an ulcerogranulomatous process. The identity of the ventricles, ventricular bands and vocal cords has been lost. There is marked thickening of the epiglottis and arytenoid folds in addition to the arytenoids.

follow the removal of tonsils which were not known to be tuberculous. This form of involvement occurs frequently as a terminal process. The larynx appears hyperemic and there are elevated, fine, granular bodies shining through the membrane. The process is widespread and rapid. The nodules are closely grouped and are of equal size, denoting the same time of deposit. Rapidly progressive ulceration occurs, which may destroy an entire section of the larynx. There appears to be a rapid fusion of the nodules which results in extensive and deep ulceration. This type of involvement can best be studied in the uvula, tonsillar pillars or palate, which may be affected at the same time as the larynx.

There is another form of miliary spread which is more benign in its local manifestations and behavior. In this form the miliary

nodules are seen in a single structure, or in two or more adjacent structures of the larynx. It may be confined to the epiglottis, or it may involve the epiglottis and its adjacent structures. At times only a ventricular band may be involved. The process is characterized by the presence of fine, granular nodules which shine through the intact mucosa. These nodules are not as closely grouped, as in the more serious form, and do not have their uniformity. The progress of the lesion is slow; in most cases ulceration does not occur, but healing takes place, leaving a marked thickening of the tissues. Ulceration may appear after some time has elapsed and is usually sluggish and indolent.

This form seems to coincide with that which most writers call lupus, for its ulcerations are painless, even when extensive. Most of the epiglottis may be destroyed without the presence of pain or any symptom, and only laryngeal examination discloses that its destruction has occurred. The thickening and scar formation are greater than that found in the average healed lesion of the larynx. The acute miliary form does not have any tendency to form fibrous tissue and heal.

Fixation of the Vocal Cord: Fixation of the vocal cord by an ankylosis of the cricoarytenoid joint occurred 28 times in this series. Both cords were involved four times. When both cords are fixed close to the midline, tracheotomy becomes necessary. For this reason involvement of the arytenoids should be watched closely for such a possibility.

Symptoms: The symptoms of tuberculosis of the larynx are extremely variable, for they depend upon a variety of pathological changes in several different locations. Symptoms may be absent even in the presence of active and extensive disease of the larynx. Occasionally the symptoms of a tuberculous lesion in the larynx may be the first indication of the presence of pulmonary tuberculosis. In this group there were 25 patients whose pulmonary condition was discovered because of their laryngeal disease. Fifty-three patients in this series had an involvement of the larynx without any symptom to indicate it.

The symptoms which are encountered in tuberculosis of the larynx are those which are known to other inflammatory diseases of the larynx. The symptoms of which this group of patients complained were distributed as follows:

TABLE VI

SYMPTOMS

Hoarseness to aphonia	856
Pain	328
Dysphagia and odynophagia	265
Dryness	102
Soreness	92
Pain without hoarseness	41
No symptoms	53
Dyspnea requiring tracheotomy	9

The following lesions cause no symptoms:

Infiltration of the epiglottis.

Edema of the epiglottis.

Tuberculoma of the epiglottis.

Ulcer of laryngeal aspect of epiglottis.

Small lesions of the interarytenoid space in the form of infiltration, edema, tuberculoma or ulceration.

Slight but uniform thickening of the vocal cords.

A small granuloma or superficial, indolent ulcer of the vocal process.

Slight to moderate infiltration or edema of the arytenoids.

Tuberculoma of an arytenoid.

Aspiration of ingested food particles or fluid occurs during the act of swallowing in the presence of extensive laryngeal disease. There are two reasons for this—the larynx may be insensitive because of the widespread ulceration and infiltration, or the glottic space cannot be completely closed because of loss of structure.

Regurgitation of fluids through the nose occurs in patients with extensive and painful disease of the larynx. The contact of the fluid with the sensitive and inflamed larynx causes a sudden spasm and closure which amounts to an expulsive effort. This sudden expulsive effort propels the fluid into the nasopharynx and out through the nose. The soft palate does not shut off the nasopharynx during the act of swallowing as is generally supposed.

Discomfort rather than pain is encountered when an infiltrative lesion or a granuloma creates the sensation of a foreign body. This is so when there is a localized increase in the volume of the vocal cord, or an infiltrative process in the region of the vocal process or the interarytenoid space. When these lesions are extensive they impart a constant sensation of the presence of a foreign body which makes the patient attempt to get rid of it by means of a nonproductive cough.

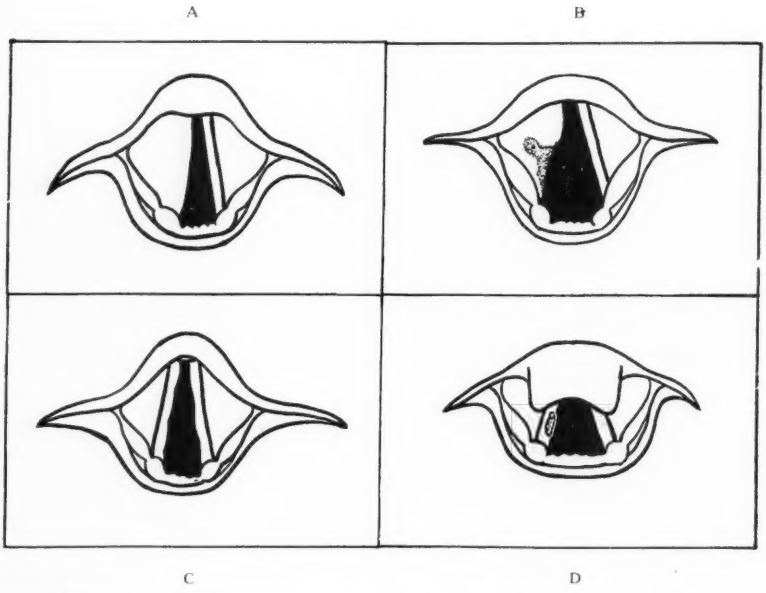


Fig. 11. Semi-diagrammatic, mirror impression of lesions causing hoarseness:
A—Swelling and edema of right ventricular band.
B—Ulceration and swelling of right ventricular band.
C—Healed and distorted vocal cords.
D—Granuloma on right vocal cord.

The impression that hoarseness is the first symptom of laryngeal disease in the tuberculous should be corrected. Hoarseness may never occur during this complication. In this series, in addition to the 53 patients without symptoms, there were 39 who suffered pain without having any hoarseness. Pain without hoarseness occurs in the presence of the following: Ulcer of the epiglottis, interarytenoid space, or of the posterior aspect of the arytenoid or cricoid cartilages, or acute perichondritis of any of the cartilages.

Dyspnea may occur suddenly or develop gradually; the latter is more frequent. It is for this reason that a patient can maintain himself on a greatly narrowed glottis which would be very serious if such a degree of narrowing had occurred suddenly. Obstruction of the laryngeal airway sufficient to cause dyspnea is due to a variety

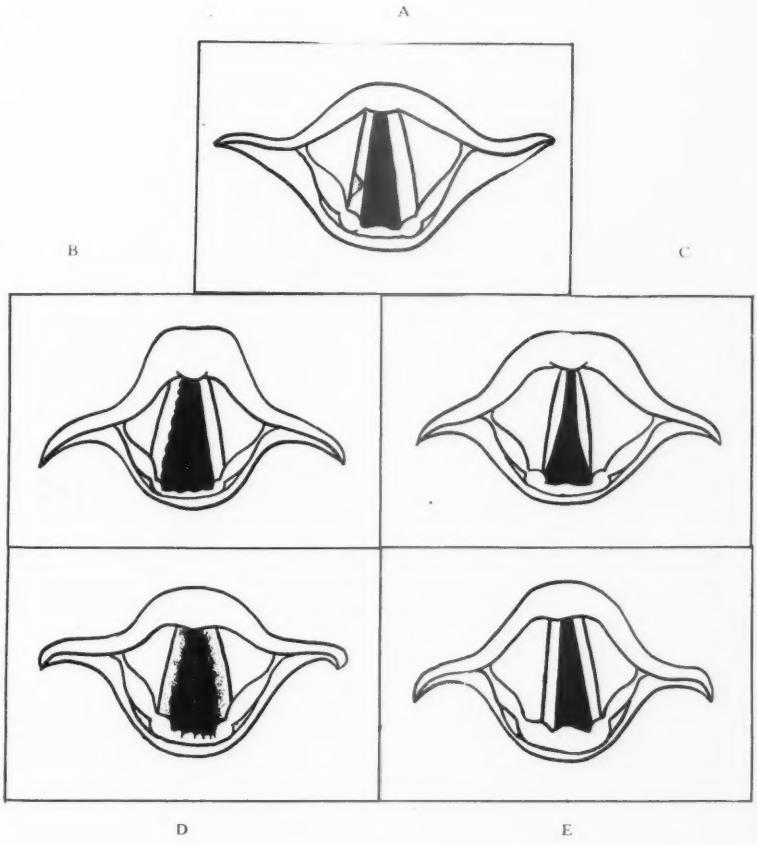


Fig. 12. Semi-diagrammatic, mirror impression of lesions causing hoarseness:

A—Granuloma of right ventricle.
B—Irregular ulceration of right vocal cord.
C—Thickening of vocal cords.
D—Granular and ulcerative vocal cords.
E—Fixation of the right vocal cord.

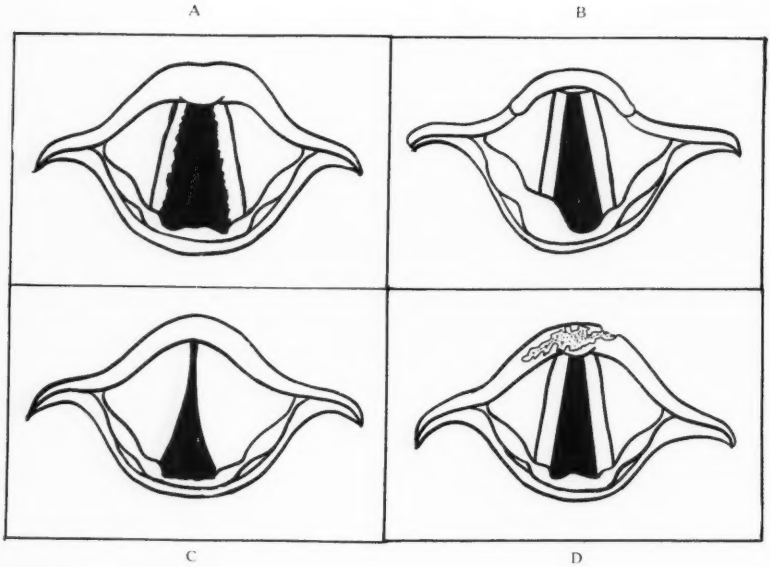


Fig. 13. Semi-diagrammatic, mirror impression of lesions causing dysphagia:
A—Ulcerations of vocal cords.
B—Acute edema of right arytenoid and aryepiglottic fold.
C—Acute edema and swelling of ventricular bands.
D—Active ulceration of epiglottis.

of causes. The most frequent of these is ankylosis of one or both cricoarytenoid joints, and the consequent fixation of the vocal cord at or near the midline. Perichondritis of the arytenoid cartilage is the most frequent cause of this fixation. Occasionally, however, perichondritis of the posterior surface of the cricoid cartilage is the primary factor. Fixation of one joint without other lesions does not create sufficient obstruction to cause dyspnea. The partial obstruction, however, causes some embarrassment when it is superimposed upon a pre-existing pulmonary dyspnea. Fixation of one cord, when associated with infiltration or tumor formation of either cord, or ventricular band or the opposite ventricle, may cause urgent dyspnea. The other lesions which cause dyspnea are of the infiltrative or granulomatous type. A frequent contributory cause is the adhesion of thick mucoid or mucopurulent secretions to extensively ulcerated areas.

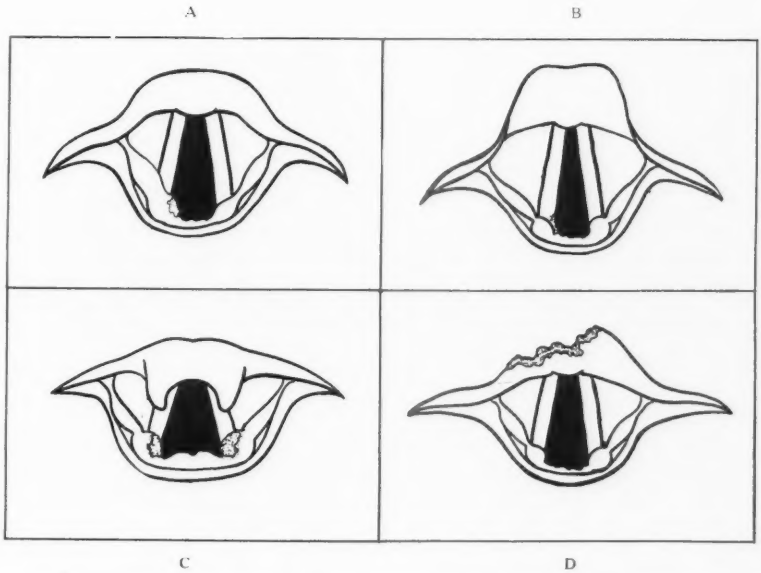


Fig. 14. Semi-diagrammatic, mirror impression of lesions causing dysphagia:
A—Ulcer of right arytenoid region.
B—Ulcer of right vocal process.
C—Ulcer of both arytenoid regions.
C—Ulceration and necrosis of epiglottis.

The subject of tuberculosis of the larynx requiring tracheotomy has been considered in a previous paper. The following indicate the type and extent of disease which required tracheotomy in some of our cases:

1. Tuberculoma of the interarytenoid region associated with necrosis of thyroid cartilage and abscess formation.

2. Edema of the arytenoids, subglottic swelling and ulceration, and a large interarytenoid granuloma.

3. Postcricoid infiltration extending upward to involve both cricoarytenoid joints.

4. Extensive ulceration of the glottic structures with considerable subglottic swelling and interarytenoid hyperplasia.

5. Edematous swelling of the epiglottis, both arytenoids and the ventricular bands, with fixation of both cords.

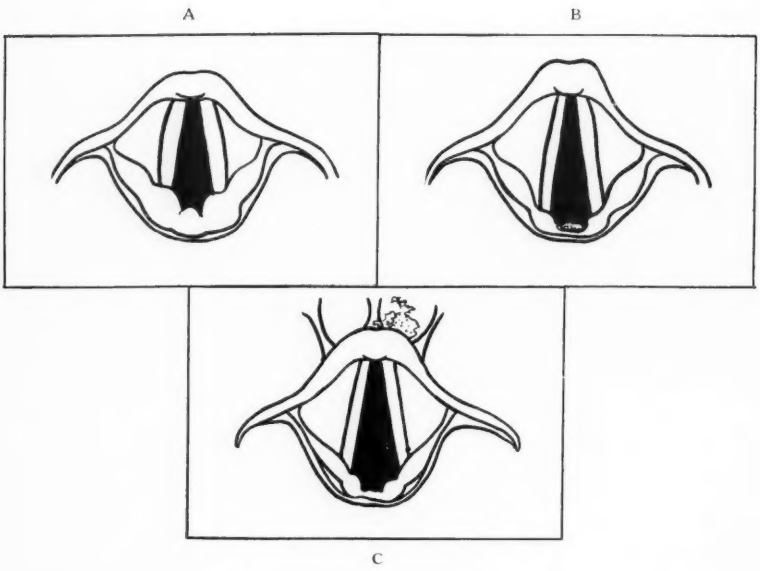


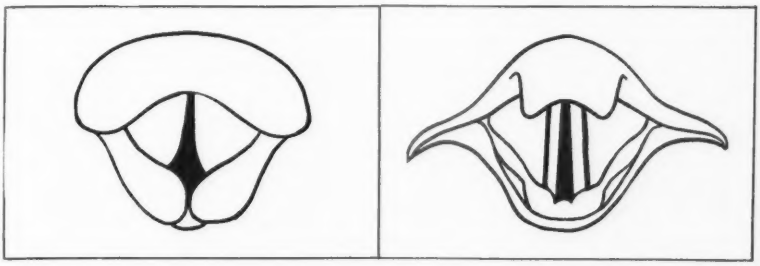
Fig. 15. Semi-diagrammatic, mirror impression of lesions causing dysphagia:
A—Acute edema of the arytenoid and interarytenoid regions.
B—Interarytenoid ulceration.
C—Ulceration of glossoepiglottic fossæ.

Dryness is not mentioned by most writers, yet it occurs quite frequently in tuberculosis of the larynx. Extensive infiltration, with or without ulceration, interferes with the mucous secretions, so that varying degrees of dryness result. This dryness may be quite annoying, and is frequently the only symptom of which many patients complain. Dryness also contributes to the production of laryngeal cough.

DIAGNOSIS

The subject of diagnosis will not be considered fully. All laryngologists are well acquainted with the diagnosis and differential diagnosis of tuberculosis of the larynx. There are, however, certain lesions in the larynx which might prove to be confusing to the best laryngologist.

There are four laryngeal pictures in the tuberculous which might confuse the laryngologist when he is examining a male over



A B

Fig. 16. Semi-diagrammatic, mirror impression of lesions causing obstruction:
A—Extensive edematous infiltration of all structures.
B—Fixation of both vocal cords due to involvement of the cricoarytenoid joints.

forty years of age: One, a small neoplasm of the vocal cord; two, a similar lesion of the ventricle; three, a subglottic infiltration; four, a uniform and symmetrical involvement of both sides of the larynx with superficial ulceration of the vocal cords, thickening and infiltration extending from the ventricular band to the subglottic region of each side. Occasionally the last picture is seen in elderly men and proves to be a scirrhous type of carcinoma. Such a lesion has been seen several times in men of 70 or over, and the diagnosis of carcinoma or tuberculosis has eventually been made in each. The importance of biopsy in these unusual cases is evident.

X-ray Diagnosis: The x-ray diagnosis of tuberculosis of the larynx is not generally accepted as a valuable aid. It appears to have some value in the hands of a few workers. The roentgenogram can add very little to that which the laryngeal mirror discloses. A lesion of the ventricle may be demonstrated on the x-ray film while it is not yet discernible by ordinary laryngeal examination. This type of lesion, however, occurs so infrequently that the value of the x-ray is negligible. In addition, Hay[8] has reported that the ventricle cannot be seen in half of the normal male larynges. The roentgenogram is of value in demonstrating the lower limit of a subglottic lesion, and as a graphic record of the laryngeal involvement.

Recently tomography, which affords a kymographic representation of the organ which is rayed, promises to furnish much more accurate information than the usual x-ray study.

Direct Laryngoscopy: It is best to rely upon the laryngeal mirror for biopsy and for a study of the laryngeal condition. In a great majority of cases of tuberculosis of the larynx the use of the laryngeal speculum should be avoided. Direct laryngoscopy is harmful and is contraindicated in the presence of an acutely active tuberculous lesion. Even a subsiding lesion might be stirred into activity by direct laryngoscopy. This has happened on more than one occasion in our experience. Direct laryngoscopy is permissible only in the chronic healing or healed stage of the disease, where there is little likelihood of activating the local lesion. This refers also to bronchoscopy.

PROGNOSIS

Analysis of a large number of cases of tuberculosis of the larynx indicates that there is no fixed rule concerning the factors and conditions which govern the prognosis of this disease. The oft-repeated statement that early diagnosis means a more favorable prognosis is open to question. It is not the time of recognition but the nature and extent of the involvement which matters. A patient may have an unrecognized tuberculoma of the interarytenoid space for a long time and his larynx heal spontaneously. On the other hand, another patient may have a generalized edematous infiltration which is recognized early and which gradually or rapidly evolves into a widespread and disintegrating ulceration, terminating fatally.

Although there are some exceptions, it is generally agreed that the laryngeal condition is correlated with and dependent upon the pulmonary disease. It is also recognized that the advent of more active and energetic treatment of the lung, both surgical and non-surgical, in recent years, has had no appreciable influence upon the prognosis of the laryngeal complication. As a result the mortality has shown a definite decline.

Levy,[11] Wood,[26] Thomson,[23] Greene,[7] Spencer,[22] Carmody[3] and others have stressed the fact that the laryngeal complication may have a harmful effect upon the pulmonary condition, just as the pulmonary disease influences the larynx unfavorably.

Analysis of Cases: An analysis of those cases which were healed and those which died has proven interesting. There were 145 healed larynges and 490 deaths among the one thousand cases studied. Of

TABLE VII

TYPE OF PULMONARY DISEASE

	Healed	Died
Bilateral	111 or 76.5%	471 or 96.2%
Caseous pneumonic	78	442
Productive	18	2
Mixed	14	18
Hematogenous	1	2
Bronchogenic	0	2
Unilateral	34 or 24.7%	19 or 3.8%
Caseous pneumonic	23	19
Productive	9	0
Mixed	2	0

TABLE VIII

SPUTUM FINDINGS

	Healed	Died
Negative	61 or 42%	8 or 1.6%
Always negative	20	
Positive, became negative	41	
Positive	84 or 58%	482 or 98.4%

the 145 healed cases, 17 died, 13 had recurrences, and 11 had an unimproved pulmonary condition. There were four pregnant women among the healed cases. Most of the patients in this series were under observation for two or more years.

There was a noticeable preponderance of infiltrative lesions among the healed cases, while among the deaths the ulcerative lesions were in the majority; the proportion of edematous lesions among the fatal cases as compared to the healed was two to one. Most of the ulcers among the healed cases were superficial.

The presence of 15 isolated lesions among the fatal cases indicates that such lesions by themselves do not offer a favorable prognosis. There is no one influencing condition, lesion or factor which bespeaks the future healing of a tuberculous larynx.

Analysis of the healed cases indicates that spontaneous healing is not rare. It also indicates that the influencing factors, such as the nature and extent of the pulmonary lesion, the nature and extent of the laryngeal disease, the presence or absence of complications, the presence or absence of a positive sputum, the successful execution of surgery or pneumothorax when indicated, are of much greater importance than any local treatment which might be used. The fact is that those lesions of the larynx which are improved by local treatment are the types which would heal spontaneously.

TABLE IX

NATURE OF LARYNGEAL INVOLVEMENT

	Healed	Died
Isolated or single lesions	28 or 20%	15 or 3%
Generalized involvement	5 or 3%	55 or 11.2%
More than one lesion	112 or 77%	420 or 85.8%

TABLE X

TYPE OF LARYNGEAL LESION

	Healed	Died
Infiltration	112 or 44.4%	223 or 30.3%
Ulcer	63 or 25.0%	275 or 34.8%
Tuberculoma	46 or 18.3%	66 or 8.9%
Edema	31 or 12.3%	189 or 26.0%

TABLE XI

PREDOMINATING LARYNGEAL LESION

	Healed	Died
Infiltration	74 or 51%	195 or 39.9%
Ulcer	30 or 19%	210 or 42.9%
Tuberculoma	23 or 18%	23 or 4.8%
Edema	18 or 12%	62 or 12.4%

It is significant that among the healed cases there were 24.7 per cent unilateral pulmonary lesions as compared with 4 per cent among the fatal cases. There were no productive lesions among the fatal cases; even among the unilateral cases there were only caseous pneumonic lesions. On the other hand, 18.6 per cent of the healed cases had productive lesions; 471, or 96.2 per cent of the fatal cases suffered with a bilateral pulmonary involvement, whereas 94 per cent of all the patients who died had active caseous pneumonic disease of the lungs.

There were 32.1 per cent among the healed cases with a negative sputum; 1 per cent of the fatal cases had a negative sputum.

In the group of healed larynges, ten patients had complications which were confined to the gastrointestinal and genito-urinary tracts. Among these there was not a single case of ulceration of the oral cavity, pharynx or tonsil, nor was there any instance of otitis media or mastoiditis. Among the deaths, however, there were a relatively

large number of these complications, which amounted to 10 per cent. The total complications among the healed cases were 10, or 6.9 per cent, as compared with 133, or 27 per cent, among the fatalities. In the fatal group the complications consisted of lesions of the gastrointestinal, genito-urinary, and upper and lower respiratory tracts, in addition to meningitis, bone involvement, hydropneumothorax, empyema, diabetes and hemorrhage.

TABLE XII

OPERATIVE PROCEDURES AMONG THE HEALED CASES

Pneumothorax (one or both)	73 or 50.0%
Thoracoplasty	14 or 9.6%
Phrenicectomy	7
Jacobeus	15

OPERATIVE PROCEDURES AMONG THE FATAL CASES

Pneumothorax (one or both)	99 or 20.6%
Thoracoplasty	23 or 4.7%
Phrenicectomy	4
Jacobeus	7

The relative number of successful pneumothoraxes is significant. The production of a successful pneumothorax usually, but not always, prevents involvement of the larynx. Several of the patients in this study developed laryngeal tuberculosis in spite of successful collapse therapy.

It is well established that, in properly selected cases, collapse therapy not only will prevent a laryngeal complication, but may also influence its healing. We can hope for improvement of the laryngeal condition only if the collapse converts an active pulmonary lesion into an inactive one.

Significance of Type of Laryngeal Lesion: It is generally accepted that lesions of the productive type indicate a more favorable prognosis. Although the presence of a tuberculoma does not insure healing, it indicates that the patient has it within his power to heal, if too many detrimental factors are not present.

An infiltrative lesion indicates a more favorable prognosis if the patient's general condition is not too bad. For this reason fixation of a cricoarytenoid joint is not to be viewed unfavorably, for it is an indication of the ability of the laryngeal tissues to undergo fibrous tissue formation.

Edematous and ulcerative lesions of the acute type are less favorable, yet they too have the ability to heal when the patient's general condition will permit.

An isolated lesion is more favorable than a generalized involvement.

It is not the early lesion or the time of recognition of involvement of the larynx which determines its future. A lesion may be very early and yet indicate a serious invasion; or a larynx may be involved for a relatively long period of time by an attenuated form of the disease. From the standpoint of future healing, it would appear that the importance of early recognition is overemphasized. This does not mean that early recognition is not an advantage in the general care of the patient, and that it should not be encouraged.

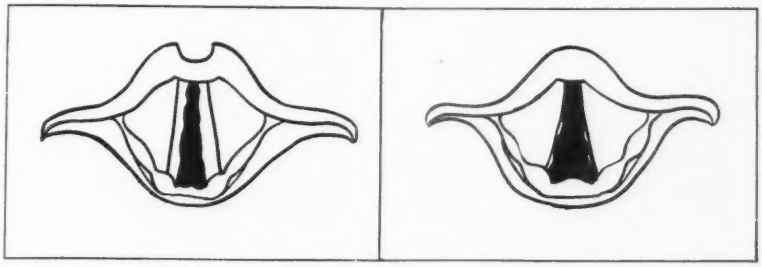
The presence of an abundance of epithelioid cells in the microscopic field indicates a fair prospect of healing, for these cells are the forerunners of fibrous tissue. If only small lymphocytes are present, this indicates either a lack of ability to form fibrous tissue, or a very early lesion in which the epithelioid cells have not yet appeared.

Pregnancy: Pregnancy usually bespeaks an unfavorable prognosis for the expectant mother with a laryngeal involvement. Many women with pulmonary tuberculosis will develop disease of the larynx during the first half of their pregnancy. Four of the healed larynges occurred among pregnant women, a fact which indicates that not all cases of laryngeal tuberculosis with pregnancy are doomed.

There are two groups of cases from the standpoint of prognosis. First, mothers who are in good condition and can be carried through their pregnancy by careful supervision; second, those with extensive pulmonary involvement, for whom abortion should be performed early. Pregnancy should never be contemplated when a laryngeal complication exists.

It would be well for the tuberculous woman not to become pregnant, for despite the fact that some get well, statistics are against them. Another consideration is the high mortality among the newborn.

Recurrences: The larynx which is healed after a tuberculous involvement cannot be considered as cured. It may be considered healed, arrested, or as rendered inert or latent. For this reason



A B

Fig. 17. Semi-diagrammatic, mirror impression of healed lesions:

A—U-shaped deformity of epiglottis, in addition to thickening and irregularity of vocal cords.

B—Arytenoid and interarytenoid thickening, in addition to loss of structure of both vocal cords.

every tuberculous larynx which has healed has the ability to become reinfected and should therefore have the benefit of a follow-up. The same factors which favor the original complication, favor a reinfection.

A frequent cause of recurrence is a lack of adherence to an orderly routine and proper regimen which has proven of value in keeping these patients well. It must be borne in mind that failure to follow instructions should not be attributed entirely to carelessness. A powerful contributory factor is the social and economic status of the individual. Many patients come to our institutions from a very unfavorable environment. It is most unfortunate that they must return to such an environment after a good deal of time, energy and money have been expended in making them well.

TREATMENT

There has never been a satisfactory treatment for all cases of tuberculosis of the larynx. The nature of the disease and its variety of lesions in several locations make it an extremely difficult therapeutic problem. The treatment of tuberculosis of the larynx must take into consideration the type of laryngeal disease and the nature of the pulmonary and general condition. A benign or inactive lesion of the larynx requires no special treatment. An active lesion

may be treated, but the treatment must take into account the degree of activity which is present.

Hundreds of medicaments and therapeutic measures have been advocated for this disease, and new ones are being introduced at frequent intervals. Surgery, which has been abandoned by most clinics, was practiced extensively during the latter part of the last century. According to Gleitzmann, surgery included anything from an incision with knives, to laryngectomy. Intubation was at one time practiced extensively for this condition. Gleitzmann's[6] article, published in 1895, included references of 227 articles dealing with surgery in tuberculosis of the larynx.

Various dyes, metallic salts, acids, powders, alkaline sprays, oils, astringents, vaccines, tuberculins, forms of light, x-ray and other therapeutic agents have been advocated as beneficial for this condition. A detailed list of the remedies which have been used and for which extravagant claims have been made would occupy a volume.

In our clinic the following measures are used: Vocal rest, oil instillations, ultraviolet radiation, electrocautery and injection of the superior laryngeal nerve and pharyngeal plexus.

Vocal Rest: Silence is prescribed for all active cases which present some expectancy of healing. Patients who have extensive ulcerative lesions and whose lungs are badly involved are not burdened with the routine of silence.

Vocal rest is not new in the treatment of laryngeal disease. In 1824, Broussais advocated absolute silence in chronic conditions of the larynx. It is very likely that silence was used by most physicians who treated laryngeal disease.

Lesions of the vocal cords, vocal processes, ventricular bands, arytenoids, and interarytenoid space, especially of the ulcerative type, are helped by vocal rest when they are not active and progressive. Complete silence is considered superior to whispered voice, which in many instances places a strain upon the patient's vocal apparatus. Whispered voice is therefore not prescribed.

Oils: All kinds of oils or combinations of oils have been used; the most popular in recent years are chaulmoogra and cod liver oils. We have studied controlled groups of patients into whose larynges these oils have been instilled, and others from whom the oil was withheld. We are convinced that neither of these oils has a curative effect. They do, however, have definite value as temporary, sooth-

ing and cleansing agents. The instillation of oil of any kind helps remove thick mucoid and mucopurulent secretions which are adherent to the laryngeal structures. We have not seen a larynx which healed while being treated with oil which would not have healed without it.

Ultraviolet Ray: The use of the Kromayer lamp with special laryngeal applicators has been a routine procedure. The value of this form of therapy lies principally in its psychological appeal. Even the very sick patients are impressed with the fact that something is being done for them when they must report to the clinic for treatment three times a week. Credit for improvement in our cases cannot be attributed to this form of therapy.

Electrocautery: The electrocautery is the best single weapon available to the laryngologist. It is excellent for reducing the size of edematous infiltrations or tuberculomata. It is also of value in searing both active and inactive ulcers so as to eliminate or reduce the pain which they cause.

When used to puncture infiltrative processes it accomplishes its purpose by creating a local reaction which brings a blood supply to an avascular field. The cautery must be applied accurately and should not be used to destroy diseased tissue. Acutely active lesions should not be treated with the cautery. It is permissible, however, to sear acutely active superficial ulcerations for the elimination of pain. Cautery puncture of the acutely inflamed structures causes ulceration and an aggravation of the disease. When cauterization of lesions on the vocal cords is contemplated, one side should be done at a sitting. A web formation may occur if both sides are done at the same time.

The cautery is usually applied to from one to four places in the larynx. It is permissible, however, to make many more applications at a single session, if the larynx is not acutely involved, and if the cauterization is carried out with due regard for possible tissue reaction. All cauterization is performed by the indirect method of laryngoscopy, after anesthetization of the larynx with a 10 per cent cocain solution. Cauterizations should be carried out at intervals so spaced as to permit a subsidence of the local reaction. A week is usually sufficient.

It is important to help these patients maintain their nutritional state; therefore, the laryngologist should resort to the injection of the superior laryngeal nerve with novocain, followed by alcohol, on the affected side whenever pain exists which he cannot control by

means of the cautery. These injections afford freedom from pain for varying periods of time. In many instances, however, the relief lasts for only a few days. Under such circumstances, section of the nerve may prove of value. In some cases the pain is caused by ulceration situated outside of the larynx, such as at the tongue base, in the pharynx or the pyriform fossa. Because of this an injection of the pharyngeal plexus with novocain is done whenever blocking of the superior laryngeal nerve is not effective. If this controls the pain, alcohol is injected into the pharyngeal plexus.

Although it is generally agreed that those cases which require injection of the superior laryngeal nerve have a poor prognosis, there are exceptions. Four of the cases which required such injections finally healed. It is interesting to note that twelve of the patients with healed larynges suffered pain.

X-ray therapy has been advocated and is being used in some clinics. Although our experience with x-ray therapy has been limited, we are impressed with the fact that those cases which react favorably would do so without any form of therapy. Acute and active cases will be made worse by x-ray therapy.

We do not use cutting instruments of any kind in tuberculosis of the larynx. Curets and forceps cause bleeding and create new avenues for the spread of the disease. We do not favor removal of the epiglottis when it is diseased. Our early experiences with epiglottidectomy were not encouraging. After this procedure there remains a stump which may prove more troublesome than the original disease. Ulcers of the epiglottis can be seared and edematous lesions punctured with the cautery.

An analysis of the 145 larynges which were healed reveals that 87 received no therapy except silence, which is routine for all cases. Nothing was used in these larynx, some of which healed, even though the patient died. This confirms the opinion I have long held, that the value of local therapy in tuberculosis of the larynx is greatly overestimated. The larynx will heal if the tissues can undergo fibrosis. If such fibrosis cannot occur, no therapeutic measure will be of value.

In conclusion, it behooves the laryngologist to understand the significance of the pulmonary disease and its many phases when he is treating laryngeal tuberculosis. With this understanding he can more properly treat the larynx and evaluate the future course and outcome of the disease.

136 East 64th Street.

BIBLIOGRAPHY

1. Andral, G.: Clinique Medicale, 1829-33.

2. Broussais, Fr. J. V.: Histoire des Phlegmasies, Paris, 1:372, 1816.

3. Carmody, T. E.: The Treatment of Laryngeal Tuberculosis. Trans. Am. Lar., Rhin. and Otol. Soc., 274, 1921.

4. Dignat, P.: Tuberculose Larynges, necrose et expulsion d'un cartilage, etc. Rev. mens. de laryngol., etc., Paris, 4:239, 1883.

5. Fletcher: Larynx and Thyroid Cartilage Perforated by Ulceration, etc. Prov. M. & S. J., London, p. 577, 1851.

6. Gleitsmann, J. W.: Surgical Treatment of Laryngeal Tuberculosis. Trans. Am. Lar. Assoc., p. 132, 1895.

7. Greene, Joseph B.: (a) Laryngeal Tuberculosis. South. M. J., 8:913, 1915. (b) Obstructive Tuberculosis of the Larynx. South. M. J., 21:341 (May), 1928. (c) Non-tuberculous Lesions of the Larynx in the Tuberculous. ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY, 38:812 (Sept.), 1929. (d) Some Aspects of Laryngeal Tuberculosis. Trans. Am. Lar., Rhin. and Otol. Soc., 1930. (e) Prognosis in Laryngeal Tuberculosis. Arch. Otolaryng., 26:18 (July), 1937

8. Hay, P. D., Jr.: The Neck. Paul B. Hoeber, N. Y., 1930.

9. Isambert: De la tuberculose miliare aigue pharyngo-laryngee. Ann. d. mal. de l'oreille et du larynx, 1:77, 1875.

10. Krause, A. K.: The Evolution of the Tubercle. Jour. of Outdoor Life, N. Y., 1927.

11. Levy, Robert: (a) Prognosis of Laryngeal Tuberculosis. N. A. M. A., (Sept. 16), 1899. (b) Prognosis of Laryngeal Tuberculosis. Laryngoscope, 14:787, 1904. (c) Laryngeal Tuberculosis. N. Y. M. J. (Sept.), 1909. (d) The Significance of Laryngeal Manifestations During the Course of Pulmonary Tuberculosis. J. Iowa St. M. Soc. (Dec. 15), 1914. (e) Laryngeal Tuberculosis. J. A. M. A. (May), 1913.

12. Lieutaud, J.: Histoire Anatomica Medica, Paris, 1767.

13. Mackenzie, M.: Diseases of the Nose and Throat. P. Blakiston, Philadelphia, 1880.

14. Monlaur, N. A. M.: De la Chondroperichondrite Tuberculose due Larynx. Thesis Paris, 293, 1912.

15. Petit, M. A.: On Laryngophthisis. Montpelier, 1790.

16. Ranke, K. E.: Primaraffekt, sekundare und tertiare Stadien der Lungentuberkulose. Deut. Arch. f. klin. Med., 119:201, 1916.

17. Raviart and Marlier: Subcutaneous Emphysema of the Face, Neck, etc., Due to a perforation of the Larynx, etc. Bull. Soc. Centre de med. du nord., 2s, 3:177, 1899.

18. Rey, P.: Case of Laryngeal Phthisis with Granulations and Tubercular Ulcerations and Perforations, etc. Progr. med., 2s, 1:355, 1885.

19. Rickmann, L.: Pathologie und Therapie der Kehlkopftuberkulose, 1930.

20. Safranek: Die Pathologie und Therapie der Kehlkopftuberkulose, etc. Mon. fur Ohrenheil., 64:635 (Nov.), 1931.

21. Schnitzler, J.: Miliary Tuberculosis of the Larynx and Pharynx. Wien. med. Presse, 22:629, 1881.

22. Spencer, Frank R.: (a) The Clinical Diagnosis of Laryngeal Tuberculosis. ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY, 32:838 (Sept.), 1923. (b) The Importance of Early Diagnosis and Treatment of Laryngeal Tuberculosis. J. A. M. A., 87:216 (July), 1926. (c) The Symptomatology, Diagnosis and Treatment of Laryngeal Tuberculosis. ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY, 37:213 (March), 1928. (d) The Diagnosis and Treatment of Laryngeal Tuberculosis. Ill. M. J. (Oct.), 1932.

23. Thomson, Sir St. Clair: The Prognostic Importance of Tuberculosis of the Larynx. Trans. A. Lar. Assoc., p. 12, 1919.

24. Tobold, A.: Clinical Diseases of the Larynx. Trans. by G. M. Beard, Wm. Wood Co., pp. 121-130, 1868.

25. Tyson, M. D.: Perforations of the Larynx from Tuberculous Laryngitis. Arch. Otolaryng., 12:297 (Sept.), 1930.

26. Wood, G. B.: Tuberculosis of the Upper Respiratory Tract. Arch. Otolaryng., 2:607 (Dec.), 1925; 11:217 (Feb.), 1930; 17:700 (May), 1933; 21:210 (Feb.), 1935; 27:476 (April), 1938.

LX

PYOGENIC MEDIASTINAL INFECTIONS:

THEIR SIGNIFICANCE TO THE OTORHINOLARYNGOLOGIST*†

ALTON OCHSNER, M.D.

AND

MICHAEL DEBAKEY, M.D.

NEW ORLEANS

Whereas otorhinolaryngologists primarily are interested in lesions affecting the upper respiratory tract and its appendages, infections of the mediastinum, although anatomically relatively distant, are of significance to them. The vast majority of mediastinal infections result from pharyngeal and esophageal injuries and gravitating infections from the neck. Because of the high incidence of neck infections in lesions of the upper respiratory tract which are commonly treated by otorhinolaryngologists and because most cervical infections are potential causes of mediastinitis, a consideration of this subject is pertinent. Although the mediastinal extension of cervical infections is extremely dangerous, the prevention of such a complication is relatively easy by the timely institution of appropriate measures. Acute mediastinitis may be classified according to various criteria, i. e., location, type of lesion, and pathogenesis. The following etiologic classification of mediastinitis is presented:

I. Injuries

  A. External
    1. Accidental
    2. Operative

  B. Internal
    1. Instrumentation
    2. Foreign bodies
    3. Peritonsillar abscess

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*From the Department of Surgery, School of Medicine, Tulane University, New Orleans, Louisiana.

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II. Extension from contiguous structures

A. Suppurative tracheo-bronchial lymphadenitis
B. Empyema
C. Pericarditis
D. Esophagitis
E. Osteomyelitis of spine, sternum, and ribs
F. Subphrenic infections

III. Neoplastic

A. Carcinoma of esophagus

IV. Metastasis

Of these various causes of mediastinitis the otorhinolaryngologist has a practical interest only in injuries and infections of the pharynx and the esophagus and tracheo-bronchial lymphadenitis. Therefore, this presentation will be limited to their consideration.

Injuries of the esophagus, either accidental or the result of instrumentation, are the most frequent causes of mediastinitis. Similarly, performance of a tracheotomy with the possible contamination of the pretracheal spaces by infected tracheal exudate is a potential danger, but rarely mediastinitis follows a tracheotomy. The relatively greater number and virulence of organisms in the esophagus than in the trachea is responsible for the higher incidence of cervical and mediastinal suppurative processes in the former than in the latter. Contained within the esophagus are large numbers of anaerobic organisms (Vincent's), which under suitable conditions (anaerobiasis) characteristically produce extensive necrosis. Even though the danger of infection with possible extension into the mediastinum is relatively slight following a tracheotomy, it is desirable in the performance of this procedure to prevent contamination of the pretracheal spaces and this probably can be accomplished best by packing the wound just below the incision.

Whether esophageal injuries occur in the cervical or in the thoracic regions makes little difference as regards their potentialities. In the former instance there is great danger of extension of infection by gravitation into the mediastinum by routes which will be discussed later. Of particular importance to otorhinolaryngologists is the possible injury to the esophagus during esophagoscopy. It is a well-known fact that the danger of injuring the esophagus is much greater during endoscopic examination than the danger of injuring the trachea and bronchi. Great care must be exercised in the introduction of an esophagoscope because of the narrowing of the upper esopha-

geal orifice reduced by the normally contracted crico-pharyngeus muscle. For this reason, injury of the posterior wall of the pharynx immediately proximal to the pharyngeo-esophageal junction is likely to occur, as has been emphasized by Jackson.[1] Injury at this site is particularly dangerous because of the marked constrictor action of the muscles of the pharynx which tend to force the pharyngeal contents into the retrovisceral space. In order to avoid the danger of such perforation during the introduction of the esophagoscope, as emphasized by Jackson,[1] the esophagoscope should lift the cricoid cartilage anteriorly to overcome the cricopharyngeal pinchcock. Obviously, in the hands of the trained esophagoscopist, perforation seldom if ever occurs, and is most likely to occur during esophagoscopy performed by the novice. Whereas perforations following esophagoscopy are most common at the pharyngo-esophageal junction, lesions resulting from bouginage occur almost invariably within the esophagus itself, either at the sites of the physiologic narrowing or in the presence of disease such as carcinoma, ulcerative processes and diverticula. Injuries of the esophagus during esophagoscopy have been reported by Myerson,[2] Friedenwald and Morrison,[3] Wiether,[4] Heatly and Pearse,[5] Wessely,[6] Piquet and Deburge,[7] and Carmody.[8] Wessely[6] reported seven esophageal injuries during esophagoscopy which occurred among 8,667 endoscopic examinations (1 in 1,238). Six of these injuries were in the esophagus and one in the pharynx. Of the seven cases, three died. We have treated two cases in which perforations of the esophagus occurred during esophagoscopy. One was admitted to the hospital within six hours after injury and had an immediate cervical mediastinostomy with recovery. The other patient was not seen until approximately forty-eight hours after injury, at which time there was an extensive phlegmonous involvement of the neck and a well-advanced mediastinitis. Although a cervical mediastinostomy was done and a considerable quantity of foul-smelling pus was evacuated from the neck and posterior mediastinum, the patient succumbed within twelve hours. The danger of esophageal perforation during bouginage is greater than during esophagoscopy, because the former is more likely to be performed blindly and is more frequently performed by inexperienced men. In contradistinction to the ease with which an esophageal bougie can be passed, esophagoscopy is more difficult and therefore is more dangerous. As a result of this, most men who attempt esophagoscopy have had training, which undoubtedly accounts for the relatively low incidence of esophageal injuries following this method of diagnosis. Because of the presence of disease in the esophagus, the possibility of false passage of a bougie through the diseased area is particularly great. This

can be obviated almost entirely by the passage of the bougie over a previously swallowed string.

Of greater importance in the production of esophageal injuries which may lead to perforation and a possible mediastinitis are perforations from ingested foreign bodies. Fifty per cent of Neuhof's[9] 66 cases of mediastinitis resulted from perforation of the esophagus by ingested foreign bodies or instrumentation, illustrating the significance of esophageal perforation as a cause of mediastinitis. In Pearse's[10] 110 collected cases, perforation of the cervical esophagus was the cause of mediastinitis in 58 per cent. Foreign bodies are less likely to produce large openings in the esophagus than is instrumentation, but are more likely to produce inflammatory lesions resulting in localized esophagitis with necrosis. As the result of extension of the localized inflammatory process into the para-esophageal tissues, mediastinitis can occur. If the inflammatory process has progressed slowly, it may become localized and even perforate into the esophagus with spontaneous cure. Because of this possibility, there are many who believe that esophageal perforations should be treated conservatively. This will be discussed more in detail under treatment.

The clinical manifestations of esophageal perforation are quite characteristic. The occurrence of spontaneous pain following esophageal instrumentation or food ingestion, particularly of fish or fowl, is of significant importance. Characteristically, the pain is greatly aggravated by swallowing and is usually limited to a segment of the esophagus. The rapidity with which the other manifestations occur depends almost entirely upon the size of the esophageal opening. In those instances in which there is gross perforation of the esophagus, particularly if this is in the upper portion, as is most frequently the case, edema and emphysema occur early and, as has been emphasized by King,[11] Heatly and Pearse,[5] Wessely,[6] Phillips,[12] Jesberg,[13] and Wangensteen,[14] can be demonstrated not only by physical but also by careful roentgenologic examination. Waldapfel[15] stated that incomplete perforations, those in which only the mucosa is ruptured, result in intramural esophageal emphysema. Whereas the emphysema is the result of introduction of air through the perforation, it may also be due to gas producing micro-organisms. In gross perforations of the esophagus there occur, in addition to emphysema, clinical manifestations of a rapidly progressive contamination and infection of the retrovisceral cervical space or the mediastinum. The clinical manifestations are largely those of systemic involvement and consist of hyperpyrexia, extreme prostration, marked leucocytosis with a disproportionate increase in the polymorphonuclear leucocyte

count, and rapid pulse; in other words, the manifestations of a profound toxemia. In contradistinction to these manifestations are the symptoms presenting in a case in which the perforation is small or in which injuries of the esophagus result in a localized esophagitis and later extension into the mediastinum. The symptoms are relatively mild, particularly the systemic ones, the principal manifestation being dysphagia. In those cases in which the lower portion of the esophagus is involved, there is usually tenderness over the spine in this area. Similarly, in instances in which injuries occur in the upper esophagus, there is tenderness at the site of injury.

There is considerable controversy among investigators concerning the type of therapy to be employed in cases of esophageal injury. Because of the great likelihood of contamination of the para-esophageal tissues with the anaerobic Vincent's organisms, neoarsphenamine in relatively large doses should be administered immediately after the diagnosis of esophageal injury has been made. Marschik[16] advocates the use of polyvalent gas bacillus serum. Because of the spontaneous subsidence of clinical manifestations in patients with severe symptoms and signs, there are many who believe that conservative therapy should be used in the majority of cases. Von Eicken,[17] because of the results which he obtained in a series of eight cases, is of the opinion that operation is not necessary even though emphysema and air in the prevertebral space can be demonstrated. He reports two cases in which recovery occurred following the use of conservative therapy. He believes that the only indication for immediate operation is rising temperature and a definite change for the worse in the patient's general condition. Myerson[2] is of the same opinion, and states "external operation may be deferred even in the presence of severe local reaction and a systemic reaction denoting absorption, fever." Others who advocate conservative therapy in esophageal perforation are Orton,[18] Clerf,[19] Lederer and Fishman,[20] Jackson[21] and Carmody.[22]

Opposed to these authorities are a large group of men who believe that immediate operation is necessary in all perforations of the esophagus. Von Hacker,[23] in 1901, suggested prophylactic "cervical mediastinotomy" and packing the wound in order to prevent the gravitation of cervical infections into the mediastinum. In 1909, Marschik and Vogel[24] emphasized the importance of prophylactic "cervical mediastinotomy" in cases of injury of the upper respiratory and alimentary tracts. On the basis of a study of 529 cases with esophageal foreign body, Schlemmer[25] advocates immediate operation in all cases of esophageal perforation in which emphysema is present.

A similar suggestion is made by Killian[26] because of his experience in 389 cases of esophageal foreign body with complications, of which 169 were operated upon. Others who believe that immediate operation is indicated are: Glogau,[27] Palmer,[28] Iglauer,[29] Hajek,[30] King,[11] Furstenberg,[31] Haslinger,[32] Phillips,[12] Heatly and Pearse,[5] Miller,[33] Churchill,[34] Lilienthal,[35] Alexander[36] and Wangensteen.[14]

We are thoroughly in accord with these latter investigators, and believe that once a diagnosis of esophageal perforation is reasonably certain, external drainage is imperative, because the danger of the performance of such a procedure is inconsequential as compared with the dangers of progression of the mediastinal infection. This is true not only of cervical perforations in which a cervical mediastinotomy with exposure of the esophagus through an incision as suggested by von Hacker[23] and popularized by Marschik[24] can be done easily, but is equally true in perforations low in the esophagus in which a posterior mediastinotomy is necessary. The administration of large doses of neoarsphenamine is imperative in cases in which mediastinostomy is used. In cases with large esophageal perforations, nothing should be taken by mouth and the esophagus placed at complete physiologic rest. Occasionally gastrostomy may be indicated.

Inflammatory lesions which give rise to mediastinitis should be divided into: (1) those which arise in the cervical region and extend to the mediastinum as gravitation infections, and (2) those which begin primarily in the mediastinum. In order to understand more clearly the routes by which gravitation infections may extend from the neck to the mediastinum, a review of the surgical anatomy of the neck is necessary. Considerable anatomical investigations concerning fascial spaces of the neck and their relationship to those of the mediastinum have been made.

According to Pearse,[10] Bichat,[37] in 1801, first attempted to study the compartments of the neck. It was not until the latter part of the last century, however, that accurate anatomic investigations concerning the cervical fasciae and the modes of extension of cervical infections to the mediastinum were studied. These were done primarily on the European continent and particularly by the Germans (Henke,[38] König and Riedel,[39] Soltman,[40] Poulsen[41] and Schmitt[42]). Relatively recently renewed interest in this subject has become evident in this country, and the anatomic investigations of Furstenberg,[31, 43] Mosher,[44] Iglauer,[45] Coller and Yglesias[46] and Pearse[10] are worthy contributions. As demonstrated by these investigators, all of the cervical structures are encased within the vaginal fascia which extends completely around the neck and is attached posteriorly to

the ligamentum nuchae. This fascia splits to enclose the sternocleidomastoid muscle on either side. The infrahyoid strap muscles are contained within a fascial compartment which extends laterally to the omohyoid muscle, above to the hyoid, and below to the clavicle. This is designated as the depressor hyoid compartment which is attached inferiorly to the posterior surface of the sternum and the clavicle. Infections anterior to this, between it and the vaginal fascia, and those within the depressor hyoid compartment which are rare, are of little significance in the production of mediastinal infection because of its inferior attachment to the sternum and clavicle, preventing extension into the mediastinum. Of great importance, however, is the visceral compartment in which are contained the cervical viscera, the esophagus, trachea, thyroid and parathyroids. This extends from the base of the skull into the mediastinum and is an important structure for the extension of infection into the thorax. According to Furstenberg,[31] the visceral space is the most important pathway between the upper cervical regions and the mediastinum. The visceral compartment is of importance in the extension of cervical infections into the mediastinum, not only within the compartment, but also in the less well defined spaces both anterior and posterior to it, the previsceral and retrovisceral spaces, respectively. The former lies between the depressor hyoid compartment anteriorly and the anterior surface of the visceral compartment posteriorly. The latter is located between the posterior surface of the visceral compartment anteriorly and the prevertebral fascia posteriorly. Injuries of the cervical esophagus and infections following thyroidectomy are likely to gain entrance to the mediastinum through the visceral compartment. In those cases in which the injury has occurred in the posterior wall of the pharynx just proximal to the pharyngoesophageal orifice, infection can gravitate into the mediastinum through the retrovisceral space behind the esophagus. In Pearse's[10] 110 cases of mediastinitis the retrovisceral space was the route of extension in 71 per cent. Retropharyngeal abscesses resulting from suppurative lymphadenitis can remain localized for relatively long periods of time but, as emphasized by Pearse, if neglected may extend through the retrovisceral space into the mediastinum.

Another important compartment is the vascular compartment in which lie the carotid artery, internal jugular vein and vagus nerve. This is a very definite compartment as is well known by every surgeon who has done any cervical dissections. Because of the presence of lymph nodes within the vascular compartment, the possibility of cervical suppuration beginning in this area is evident. As Pearse[10] has emphasized, the vascular sheath is of importance not only because

of the possibility of gravitation within it, but also because of extension into the mediastinum along the sheath. The vascular sheath is of particular importance to otorhinolaryngologists because of the possibility of extension of infection from the parapharyngeal space. As emphasized by Pearse, this space may be contaminated by needle puncture during the performance of local analgesia for tonsillectomy. Also peritonsillar, retropharyngeal, and parotid abscesses may rupture into it.

The clinical manifestations of the cervical inflammatory processes which may, by gravitation, result in mediastinitis vary considerably according to the type of the originating lesion. In those cases in which there has been an extension from an inflammatory process within the pharynx, the symptoms of the cervical infection are antedated by those of the originating lesions, such as tonsillitis, pharyngitis, etc. Pain in the region of the inflammatory process is the most constant symptom. This is aggravated by movement of the neck, particularly extension, and is usually aggravated by deglutition. The pain, according to Sauerbruch,[47] is frequently increased during inspiration and with each systolic contraction of the heart. Cough is a prominent manifestation and is frequently harassing in character. Dyspnea, particularly in the fulminating cases, may be quite marked. Because of irritation of the phrenic nerve by the inflammatory process, hiccough may occur and in advanced cases, paralysis of the diaphragm may be present. In addition to these symptoms and signs, there are the systemic manifestations of infection, such as fever and leucocytosis. Extension of a cervical infection into the mediastinum is frequently associated with marked tenderness in the suprasternal fossa, and particularly on moving the trachea in various directions. In such cases, careful examination of the pharynx is of utmost importance in order to detect if possible a retropharyngeal abscess which can be drained transpharyngeally before extension has occurred. In instances in which infection has already extended into the mediastinum, retrosternal pain and tenderness over the sternum and adjacent costal cartilages are of diagnostic importance. Occasionally there is tenderness along the spine, particularly in those cases in which the infection has extended below the fourth dorsal vertebra. Edema and redness in the suprasternal and supraclavicular fossae may be present. In those cases in which there is considerable increase in mediastinal pressure, dilatation of the superficial veins of the neck, thorax and upper extremities may occur. There is usually increase in the retrosternal dullness, particularly in cases in which there has been gravitation of the infection from the neck. In the progressive form, not infrequently a peculiar mill-wheel sound is audible over the

sternum, which, according to Sauerbruch,[47] is due to movement of the heart and large vessels in the fluid-soaked mediastinal tissues. This sound is not unlike that heard in emphysema of the mediastinum, in pneumopericardium, and in embolism of the heart.

Of great importance in the diagnosis of mediastinal infections are the roentgenologic findings. As emphasized above, in perforations of the posterior wall of the pharynx and the esophagus, the visualization of air in the retrovisceral space is of diagnostic significance. Not infrequently this infiltration of air extends into the posterior mediastinum. Because of extension into the retrovisceral space the trachea is usually displaced anteriorly. Widening of the mediastinal shadow in anterior-posterior roentgenogram is of diagnostic importance. Of great diagnostic value roentgenologically is the sharp outline of the shadow produced by infection. Assmann and Lenk[48] stated that in diffuse exudative mediastinitis the lateral borders of the mediastinum become more or less vertical. Pohl,[49] Kornblum and Osmond[50] have stressed the importance of taking roentgenograms in the upright position. As emphasized in previous publications,[51] oblique and stereoroentgenograms should never be omitted in suspected cases of mediastinitis, because, frequently, only from these views can a positive diagnosis be made.

The treatment of mediastinitis resulting from gravitation of cervical infections consists primarily of prophylaxis. By appropriately treating the originating infection and placing the patient in the head-low (Trendelenburg) position, gravitation of these infections into the mediastinum can be prevented. In those cases in which infection seems imminent or in which extension into the mediastinum has already occurred, cervical mediastinostomy, as first suggested by von Hacker[23] and popularized by Marschik,[24] should be done. This is performed with the patient in the extreme Trendelenburg position. An incision is made along the anterior border of the sternocleidomastoid muscle, preferably on the right side, which is the usual site of involvement because the right compartment of the suprapericardial mediastinum contains more lymphatics than the left (Furstenberg[31]). (Fig. 1-A.) There is also less danger of opening the pleura on the right, because on this side it is in less intimate contact with the esophagus and the suprapericardial portion of the mediastinum than on the left. The lateral wall of the esophagus and trachea are exposed by laterally retracting the infrahyoid muscles and the carotid sheath with its contents (Fig. 1-B and C). By blunt dissection downward into the mediastinum, infections in the superior portion can be drained. In those cases in which the opera-

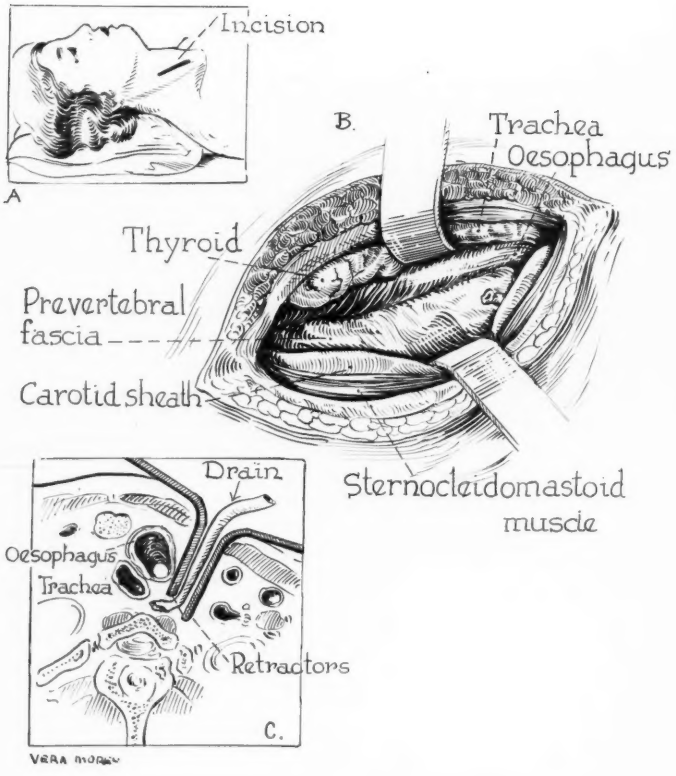

Fig. 1. Technique of cervical mediastinostomy. (A) Skin incision is made along anterior border of right sternocleidomastoid muscle in its lower portion. (B) Carotid sheath and sternocleidomastoid muscle are retracted laterally, whereas thyroid and trachea are displaced medially, exposing esophagus. It may be necessary to ligate inferior thyroid vein. (C) Cross section of neck, illustrating approach to and drainage of retrovisceral space.

tion is done prophylactically it is possible to dissect posteriorly behind the esophagus in the retrovisceral space and wall off this area by means of gauze (Fig. 1-C). Neumann[52] suggests the simultaneous ligation of the homolateral internal jugular vein, especially in the presence of chills. Postoperatively, the patient should be kept in the head-low (Trendelenburg) position to facilitate drainage by gravitation. We agree with Pearse[10, 53] that it is not necessary to make the incision throughout the entire length of the sternocleidomastoid muscle, but that a much shorter incision will suffice.

Although, as mentioned above, mediastinitis can occur in a number of different ways aside from extension from the cervical region, only those which in the tracheo-bronchial lymph nodes are of interest to otorhinolaryngologists will be considered in this presentation. Although these infections originate in the thorax, they are of significance to the bronchoscopist because of the possibility of bronchoscopic recognition and drainage. Such suppurative lesions occurred in 13 cases (11.8 per cent) of Pearse's[10] series. In previous publications,[51] the importance of acute mediastinal lymphadenitis as the cause of mediastinum has not been or still is not sufficiently appreciated because of the infrequency of correct diagnoses. It is a well-known fact that inflammation of the regional lymph nodes usually accompanies inflammatory processes in various portions of the body. Although a physician is likely to examine for evidence of cervical lymphadenitis in upper respiratory tract infections, he is likely not to consider the possibility of a mediastinal lymphadenitis in lower respiratory tract infections. Undoubtedly, in the mediastinal lymph nodes, as elsewhere in the body, inflammatory processes subside spontaneously without progressing to suppuration, and it is possible that the diagnosis of acute mediastinal lymphadenitis is made only when suppuration occurs, and then it is frequently overlooked. Mediastinal lymph nodes are infected often because at no time is the respiratory tract, including the trachea, the bronchi and lungs, free from bacteria. Eloesser[54] states: "The mediastinum is a cesspool where products of all inflammations that beset the chest and some of the abdominal ones gather." Mediastinal lymphadenitis is never primary but is always secondary to some inflammatory process elsewhere. Pyogenic infections of the lung, the bronchi and the trachea are the most frequent causes. More rarely infections of the esophagus, the pleura, the pericardium and the thoracic wall may be antecedent lesions. Streptococci are the usual offending bacteria, as they more often invade the lymphatic system than other micro-organisms. This presumption is substantiated by the observations of Hess,[55] who in thirty-six autopsies found streptococci in eight (22 per cent), staphy-

lococcus aureus and B. pseudodiphtheria in two, B. coli and various sarcinae in five, B. subtilis in one and pneumococci in one. In a previous publication,[51] the anatomic arrangement of the bronchial and tracheal lymph nodes is described. This is based largely upon the anatomic investigations of Sukiennikow.[56] There are three groups, one located below the bifurcation and one each on the right and left sides of the trachea. The inferior tracheo-bronchial space contains from nine to twelve lymph nodes and is bounded posteriorly by the esophagus on the left, by the dorsal vertebra, vessels and nerves on the right, and anteriorly by the pericardium. Laterally and above it is bounded by the two main bronchi and the bifurcation of the trachea. Below, this space extends to the level of the pulmonary veins and to the left auricle. The left tracheo-bronchial space contains from three to six lymph nodes. It is bounded posteriorly by the esophagus and the descending aorta, on the left by the ductus arteriosus, below by the left tracheo-bronchial angle, above by the aortic arch, on the right by the trachea. The right tracheo-bronchus space contains from five to seven lymph nodes. It is bounded below by the right main bronchus and the right pulmonary artery, on the left by the trachea and the ascending aorta, on the right by the mediastinal pleura and the azygos vein, and anteriorly by the superior vena cava. In addition to these, Delamere[57] described a group of glands located in the anterior mediastinum consisting of from four to six glands anterior to the transverse arch of the aorta, and another group in the posterior mediastinum in the region of the esophagus behind the pericardium.

Lerche[58] has emphasized the importance of infection of the tracheo-bronchial lymph nodes as a cause of symptoms referable to the thorax. The symptoms resulting from acute pyogenic infection of mediastinal lymph nodes are largely of two types: (1) those with systemic manifestations of infection, such as fever, chills and leucocytosis, and (2) those produced mechanically by enlarged nodes compressing the trachea and bronchi. Because of the inflammatory process, acute tracheitis and bronchitis occur with manifesting bronchorrhea, cough and expectoration. A substernal or interscapular pain associated with dysphagia, especially during the course of an acute respiratory tract infection, suggests mediastinal lymphadenitis. In children the mechanical compression of the trachea and bronchi may cause cyanosis, wheezing and marked dyspnea.

Relatively rarely, an infection will not remain confined to the lymph nodes but will extend to the mediastinal areolar tissue with the production of a progressive mediastinitis. Suppuration of the

glands with rupture into the bronchus or into the mediastinum can occur. Lerche[59] emphasized the possibility of rupture of a suppurating tracheo-bronchial lymph node into the trachea in pyogenic as well as in tuberculous infections. In addition to rupture into the mediastinum or into the trachea and bronchi, lymphadenitis is of importance because of secondary changes which may occur in the thorax. These consist of distortion and kinking of the bronchi, bronchiectasis, diverticulum, perforation of the esophagus, and tracheo-esophageal fistula. Lerche[58] is of the opinion that large mediastinal lymph nodes are of importance in the production of bronchiectasis in children in one of two ways: (1) by producing obstruction to the bronchi and trachea, and (2) by interfering with the lymph drainage from the lung and bronchi, because of the "blockage" of the lymph channels in the nodes as the result of antecedent infections such as measles or influenza. The latter, in our opinion, is the more important.

The clinical manifestations of such a case consists of the pulmonary and systemic symptoms of continued infection and the persistence of tenderness along the spinal column in the region of the tracheal bifurcation. Rarely there may be an increase in the interscapular dullness.

Unless the possibility of acute mediastinal lymphadenitis is considered by the clinician, the diagnosis is usually not made, as the clinical manifestations of the primary lesion (pulmonary) will overshadow those of the lymphadenitis. The persistence of the systemic manifestations after the primary lesion has subsided should make one suspicious of the development of a suppurative mediastinal lymphadenitis. In a patient in whom such symptoms do persist, careful roentgenographic study should be made in order to determine the presence or absence of enlarged hilar shadows. The use of serial roentgenograms is of inestimable value. Whereas the persistence of systemic signs of infection is usually suggestive of empyema in a patient who has had an antecedent pulmonary infection, because this is one of the most frequent complications of such an infection, the absence of pleural involvement should immediately suggest the possibility of a suppurating lymphadenitis. This is well illustrated by a case which we have had. A boy, twelve years of age, who had had an antecedent pneumonia of the left lower lobe, continued for five weeks to have fever and leucocytosis although the primary pulmonary infection subsided very satisfactorily. Roentgenographic examinations from the beginning showed an increase in the hilar shadow on the left side which persisted throughout the entire illness (Figs. 2 and

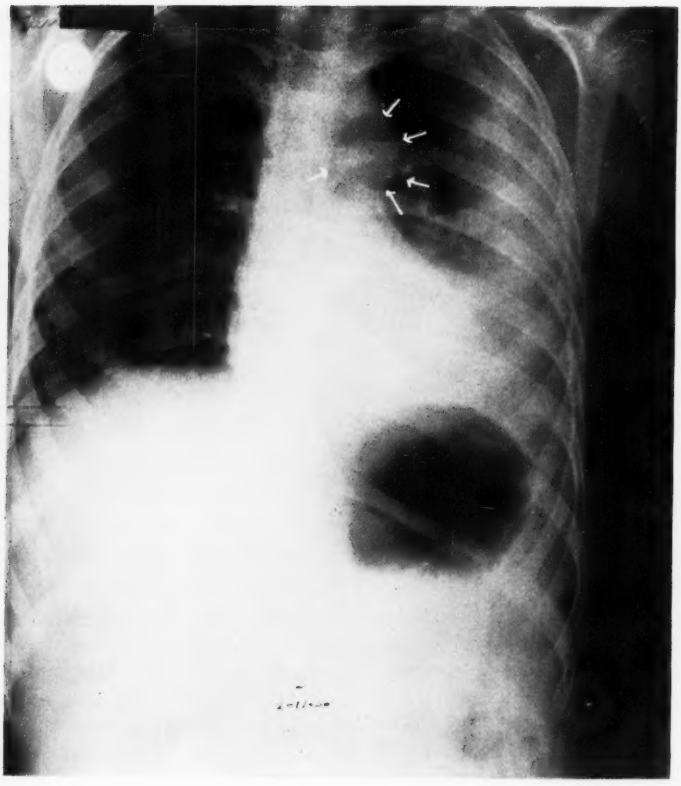

Fig. 2. Antero-posterior roentgenogram of thorax, showing mediastinal bulging in left hilar region with compression of left main stem bronchus.

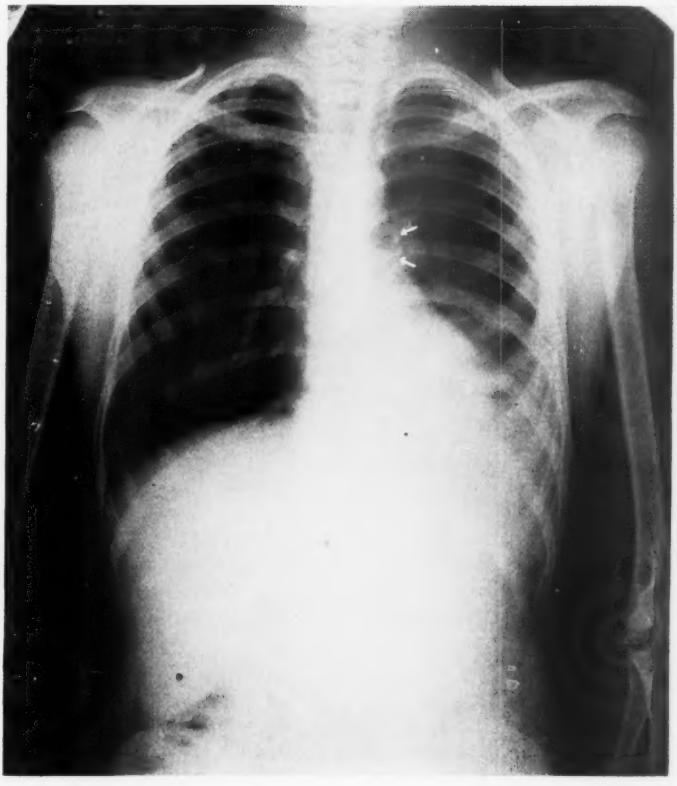

Fig. 3. Antero-posterior roentgenogram of thorax of same patient shown in Fig 2 following extrapleural drainage of mediastinal abscess. Arrows indicate site of paravertebral resection of eighth rib.

3). Ordinarily one would have suspected the possibility of the development of an empyema with persistence of these manifestations, but because this could be definitely ruled out by physical and roentgenographic findings and because of the persistence of the hilar shadow on the left side, a diagnosis of suppurative mediastinal lymphadenitis was made. In order to determine whether the process was pointing into the trachea or bronchi, bronchoscopy was performed by Dr. LeJeune. The opening of the left main stem bronchus was narrowed apparently from extrinsic pressure on the anterior and inferior walls. There was no evidence of pointing. Immediately following bronchoscopy an aspirating needle was introduced in the eighth intercostal space about four cm. to the left of the midline and directed anteriorly and medially. Twelve cubic centimeters of thick pus was aspirated. Subsequent to this the patient's temperature, which had been ranging from 102 to 103.6 degrees, fell to normal and remained there for twenty-four hours. With the reappearance of fever it was decided that the abscess should be drained by means of a posterior mediastinostomy. Under gas oxygen anesthesia, a paravertebral longitudinal incision was made over the eighth rib and its paravertebral portion was resected (Fig. 4). The mediastinal pleura was dissected from the antero-lateral surface of the body of the vertebra, exposing in the mediastinum the abscess which measured 4 x 4 x 1 cm. This was drained, following which the patient made an uneventful recovery.

As most cases of acute mediastinal lymphadenitis subside spontaneously, even without a diagnosis having been made because the condition is not suspected, the prognosis is good. Only in those instances in which suppuration occurs or in which the infection, because of it virulence, does not remain localized in the lymph nodes, is the prognosis grave.

The treatment of acute mediastinal lymphadenitis, because the condition usually subsides spontaneously, is essentially conservative and symptomatic. If the symptoms (dysphagia, cyanosis and substernal pain) referable to the mediastinum persist, bronchoscopy should be performed. Lerche[59] recommends and has successfully performed bronchoscopic drainage in both pyogenic and tuberculous adenitis. As mentioned in the above described case, bronchoscopy was done as a diagnostic procedure, but because of the absence of pointing into the bronchus, bronchoscopic drainage was not performed. By prompt bronchoscopic drainage in those cases in which there is pointing into the tracheo-bronchial tree, the danger of aspiration of necrotic material can be obviated and frequently a perfora-

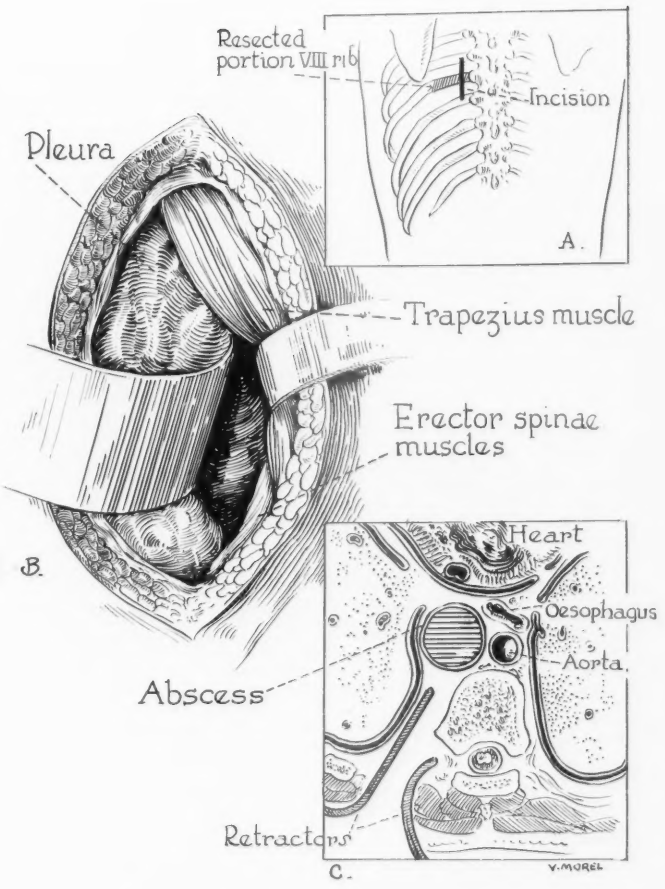

Fig. 4. Technique of posterior mediastinostomy. Paravertebral longitudinal. (A) Skin incision over eighth rib approximately 6 to 8 cm. in length. Resection of paravertebral portion of rib as represented in drawing by shaded area. Obviously the rib selected for resection should be opposite the abscess. Thus, in some cases, it is preferable to resect the seventh or the ninth ribs. (B) Trapezius and erector spinal muscles retracted medially. Parietal pleura separated from lateral aspect of bodies of vertebrae and mobilized laterally, exposing posterior mediastinum. (C) Cross section of thorax showing approach to abscess in posterior mediastinum and possibility of extrapleural drainage.

tion of the abscess into the mediastinum can be prevented. Ideally, if drainage can be secured into the trachea and bronchi and immediate removal of the purulent material accomplished, a cure should result without any further extension. However, in those cases in which suppurating mediastinal lymph nodes cannot be successfully drained bronchoscopically, external drainage by means of posterior mediastinostomy should be done (Fig. 4). Such a procedure can be performed easily and safely, and one need not fear opening the mediastinum in a case in which there is a suspected mediastinitis. Because of the fact that most cases of mediastinal lymphadenitis subside spontaneously, do not progress to suppuration, or do not extend into the mediastinum thus obviating the necessity of external drainage, it is the general impression that posterior mediastinostomy should seldom if ever be done. Whereas posterior mediastinostomy is indicated relatively infrequently because suppuration is uncommon, when this eventuality does occur, no time should be lost in performing the procedure.

The necessity of early drainage of the posterior mediastinum is particularly great in those cases of foreign body perforations of the lower esophagus. Because of the resulting mediastinal infection and its rapid extension, prompt institution of posterior paravertebral drainage is the only method which will save the life of the patient. This is particularly important, because most of these infections are anaerobic, and the institution of drainage not only permits the evacuation of its contents but also produces aerobiasis. In cases in which there is infection with anaerobic organisms, irrigation of the wound with oxidizing agents is important. As emphasized above, in cases with esophageal perforation, the placing of the esophagus at complete rest is imperative. Occasionally gastrostomy may be necessary. Because many of these infections are caused by anaerobic Vincent's organisms, the normal inhabitants of the mouth, the administration of neoarsphenamine in large doses is frequently life-saving.

It is our opinion that in those cases in which there is injury of the distal portion of the esophagus even after the performance of a gastrostomy, the danger of extension of infection from the swallowing of mouth secretions is great. This, we believe, can be obviated by the introduction of a small nasal tube down to or just above the site of perforation. The tube should have several openings and constant suction applied. In this way, the mouth secretions and the danger of forcing these into para-esophageal tissues can be prevented.

SUMMARY

1. Mediastinitis frequently is a complication of injuries to the esophagus and less frequently follows tracheal injuries.

2. Instrumentation (esophagoscopy and bouginage) and foreign bodies are the most frequent causes of esophageal injury.

3. Anaerobic infections usually follow esophageal injury.

4. Whereas many cases of esophageal injury subside spontaneously, in most instances external drainage is indicated.

5. Cervical infections may extend to mediastinum along definite fascial pathways.

6. The prompt institution of appropriate therapy of the underlying infection will prevent extension to the mediastinum.

7. Prophylactic cervical mediastinostomy is indicated in progressive infections.

8. Tracheo-bronchial lymphadenitis is associated with all intrathoracic pyogenic infections.

9. Relatively infrequently suppuration in the tracheo-bronchial lymph nodes occurs with possible rupture into a bronchus or mediastinum.

10. Bronchoscopic examination is indicated in these cases and aspiration should be done when there is pointing.

11. Posterior mediastinostomy should be done if bronchoscopic evacuation is not possible.

1430 TULANE AVENUE.

REFERENCES

1. Jackson, Chevalier, and Jackson, Chevalier L.: Bronchoscopy, Esophagoscopy and Gastroscopy. W. B. Saunders, Philadelphia, 1934.

2. Myerson, M. C.: Perforations of the Esophagus. ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY, 37:545, 1928.

3. Friedenwald, Julius, and Morrison, Theodore H.: Perforation of the Esophagus Due to Instrumentation (with a report of three cases). Am. J. Med. Sc., 167:194, 1924.

4. Wiether, C.: Ueber Oesophagusperforationen, bervorgerufen durch Oesophagoskopie. Ztschr. f. Hals- Nasen- u. Ohrenh., 28:58, 1930.

5. Heatly, C. A., and Pearse, H. E., Jr.: The Management of Perforations of the Cervical Esophagus. ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY, 41:1235, 1932.

6. Wessely, E.: Zur Frage der kollaren Mediastinotomie. Monatsch. f. Ohrenh., 69:69, 1935.

7. Piquet, J., and Deburge, A.: Les Perforations Récentes de l'oesophage cervical: Indications et Technique de la Mediastinotomie cervicale Précose. Ann. d'otolaryngol., pp. 919-930 (Aug.), 1935.

8. Carmody, T. E.: Esophagoscopy Accidents. ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY, 44:1162, 1935.

9. Neuhof, H.: Acute Mediastinal Infections, with Special Reference to Suppuration. J. Thoracic Surg., 6:184, 1936.

10. Pearse, Herman E.: Mediastinitis Following Cervical Suppuration. Ann. Surg., 108:588, 1938.

11. King, E.: Perforation of the Esophagus with Report of Six Cases. ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY, 38:351, 1929.

12. Phillips, C. E.: Mediastinal Infection from Esophageal Perforation. J. A. M. A., 111:998, 1938.

13. Jesberg, Simon: Discussion of Phillips' paper, Ref. No. 12.

14. Wangensteen, O. H.: Discussion of Pearse's paper, Ref. No. 10.

15. Waldapfel, R.: Discussion of Wessely's paper, Ref. No. 6.

16. Marschik, H.: Discussion of Wessely's paper, Ref. No. 6.

17. Von Eicken: Zur Frage der verletzung des Hypopharynx und des obersten Oesophagus abschnittes durch Fremdkörfer. Ztsch. f. Hals- Nasen- u. Ohrenh., 15:387, 1926.

18. Orton, B. B.: Mediastinitis Following Esophageal Foreign Body: Cases. Arch. Otolaryng., 12:635, 1930.

19. Clerf: Discussion of Orton's paper, Ref. No. 18.

20. Lederer, F. L., and Fishman, L. Z.: Prophylactic Mediastinotomy for Perforating Esophageal Foreign Bodies: Report of Unusual Cases. Arch. Otolaryng., 19:426, 1934.

21. Jackson, Chevalier L.: Quoted by Lederer and Fishman, Ref. No. 20.

22. Carmody: Discussion of Orton's paper, Ref. No. 18.

23. Von Hacker: Zur Operativen Behandlung der Perioesophagealen und Mediastinalen Phlegmone nebst Bemerkungen zur Technik der Collaren und Dorsalen Mediastinotomie. Arch. f. klin. Chir., 64:478, 1901.

24. Marschik, H., and Vogel, H.: Fremdkörfer in dem oberen Luft- und Speisewegen mit besonderer Berücksichtigung der Oesophagotomie. Wien. klin. Wchnschr., 22:1405, 1909.

25. Schlemmer, F.: Erfahrungen mit Oesophagus Fremdkörpern in einem Zeitraume von zehn Jahren. Arch. f. klin. Chir., 114:37, 269, 1920.

26. Killian, H.: Beitrag zur operativen Behandlung komplizierte Fälle von Fremdkörpern des Speiserohre mit besonderer Berücksichtigung der Indikationen. Arch. f. klin. Chir., 122:382, 1923.

27. Glogau, O.: A Typical External Operation for Abscesses Descending from the Upper Air Passages and the Base of the Tongue. Laryngoscope, 39:529, 1922.

28. Palmer, F. E.: Descending Abscesses in the Neck. J. A. M. A., 83:2067, 1924.

29. Iglauer, S., and Ransohoff, J. L.: Perforation of the Esophagus by a Foreign Body, with Report of a Case Presenting Unusual X-ray Signs. Laryngoscope, 34:821, 1923.

30. Hajek: Discussion of von Eicken's paper, Ztschr. f. Hals- Nasen- u. Ohrenh., 15:3921, 1926, Ref. No. 17.

31. Furstenberg, A. C.: Acute Suppuration of the Mediastinum. Tr. Am. Laryng., Rhin. and Otol. Soc., 35:210, 1929.

32. Haslinger, F.: Quoted by Lederer and Fishman, Ref. No. 20.

33. Miller, J. W.: Extraction of Foreign Bodies from the Food and Air Passages by Direct Methods. Arch. Otolaryng., 16:196, 1932.

34. Churchill, E. D.: Discussion of Pearse's paper, Ref. No. 10.

35. Lilienthal, Howard: Discussion of Pearse's paper, Ref. No. 10.

36. Alexander, John: Discussion of Pearse's paper, Ref. No. 10.

37. Bichat, X.: Anatomie Générale, Paris, Brasson, Gabon, and Cie, 1801.

38. Henke, P. J. W.: Beitrage zur Anatomie des Menschen mit Beziehung auf Bewegung. C. F. Winter, Leipzig, 1872.

39. König, F., and Riedel, B.: Die entzundlichen Processe an Hals und die Gerschwulst an Hals. Deutsch. Chir., 36, 1882.

40. Soltman, O.: Die Ansbreitungsbezirke der Congestionsabscesse bei der Spondylarthorace der Kinder. Jahrb. f. Kinderh., 7:267, 1874.

41. Poulsen, K.: Ueber die Fascien und die Interfascialen Räume des Halses. Ztschr. f. Chir., 23:223, 1886.

42. Schmitt, Adolphe: Die Fascienscheiden und ihre Beziehungen zur Senkung Abscessen. J. F. Lehmann, Munchen, 1893.

43. Furstenberg, A. C., and Yglesis, L.: Mediastinitis: Clinical Study with Practical Anatomic Considerations of Neck and Mediastinum. Arch. Otolaryngol., 25:539, 1937.

44. Mosher, H. P.: The Submaxillary Fossa Approach to Deep Pus in the Neck. Tr. Am. Acad. Ophthalmol., 34:19, 1929.

45. Iglauer, S.: Surgical Approaches to Deep Suppuration in the Neck and Posterior Mediastinum. Arch. Otolaryng., 21:707, 1935.

46. Coller, F. A., and Yglesias, Luis: The Relation of the Spread of Infection to Fascial Planes in the Neck and Thorax. Surgery, 1:323, 1937.

47. Sauerbruch: Chirurgie die Brustorganie. Julius Springer, Berlin, 1925.

48. Assmann and Lenk: Quoted by Pohl, Ref. No. 49.

49. Pohl, R.: Zur Diagnose der citrigen Mediastinitis. Med. klin., 31:1106, 1935.

50. Kornblum, K., and Osmond, L. H.: Mediastinitis. Am. J. Roentgenol., 32:23, 1934.

51. Ochsner, Alton: Surgery of the Thorax. Brennemann's Pediatrics, Vol. II, Chap. 54, W. F. Prior, Hagerstown, 1936.

52. Neumann, H.: Discussion of Wessely's paper, Ref. No. 6.

53. Pearse, Herman E., Jr.: The Operation for Perforation of the Cervical Esophagus. S., G. and O., 56:192, 1933.

54. Eloesser, L.: Bronchial Stenosis. J. Thoracic Surg., 1:194, 1931.

55. Hess, L.: Zur Frage der latenter Mikrobismus. Zentralbl. f. Bakteriol., 44:11, 1907.

56. Sukiennikow, W.: Topographische Anatomie der bronchialen und trachealen Lymphdrüsen. Berlin klin. Wchnschr., 40:316, 347, 369, 1903.

57. Delamere: Cited by Rosenfeld and McCauley. Northwest Med., 28:199, 1929.

58. Lerche, W.: Infections of the Lymph Nodes of Bronchial Tree. Arch. Surg., 16:338, 1928.

59. Lerche, W.: Infected Mediastinal Lymph Nodes as Source of Mediastinitis. Arch. Surg., 14:285, 1927.

LXI

SINUSITIS: THE PRESENT RATIONALE OF TREATMENT*

CHARLES T. PORTER, M.D.

BOSTON, MASS.

There is very little which is new concerning the operative technique in the treatment of sinus disease, and I shall not attempt any detailed description of it here. The allergist has made and is still making progress, and chemotherapy is becoming more and more valuable as an adjunct to our treatment. In many cases, however, nothing can supplant good surgery. In New England, where sinus disease is very prevalent, the most difficult problem of the otolaryngologist today is, how to overcome the prejudice of the general practitioner and the public to surgical treatment. We, as specialists, are in a large measure responsible, partly on account of poor technical surgery and partly because of lack of good judgment in the matter of knowing when to operate. We are advised by some writers on sinusitis never to operate during the acute stage. It seems this is as dangerous as to say always operate. One must evaluate the various conditions existing and use one's best judgment. If this is good, the outcome is apt to be good, if not, it is the reverse.

In acute conditions the primary object is to secure drainage and abatement of inflammatory reaction. Immediate surgery is not always advisable. I have been able to obtain complete success by conservative methods in many acute cases. This consists of rest, warm, even temperature with plenty of moisture in the air, and daily shrinkage with a weak solution of cocain followed by warm saline irrigations. When this treatment has reduced the inflammatory process to a subacute state where drainage is well established and the temperature has been normal for some time, I prefer, whenever possible, to send the patient to a warmer climate. This invariably hastens recovery and insures against relapse.

In the fulminating type of infection, immediate surgery is often necessary. In my experience this surgery should be extensive enough to obtain immediate and adequate drainage of all the sinuses involved. It is here that the most serious complications arise, such

*Read at the annual meeting of the American Laryngological Association, May 24, 1939, at Rye, N. Y.

as orbital infections, extradural and subdural abscess, with subsequent brain abscess, meningitis, and osteomyelitis. It is often advisable to remove not only the front wall of the frontal sinus but the posterior wall as well, along with the floor, and do a complete exenteration of the ethmoid. The gross appearance of the posterior wall of the frontal sinus gives no evidence as to what is behind it. Nor is the x-ray of any help in the early stages. I am convinced that this radical procedure should be followed in all cases where there is an increase in the cell count in the spinal fluid or any signs of meningeal irritation. These wounds may have to be left open for weeks or even months.

In osteomyelitis resulting from an infection of the frontal sinus, I follow the method advocated by Dr. Mosher, and remove the entire bone in one section, endeavoring to include the whole of the sinus and sufficient bone to give a wide margin beyond the area of evident infection. It is a great temptation to be less radical in the hope that we are dealing with a simple bone necrosis, only to find later that we have lost the best opportunity to get ahead of the disease. At the Massachusetts Eye and Ear Infirmary we have demonstrated that to wait for x-ray evidence of extension is frequently disastrous, as the disease is from one to two weeks ahead of the x-ray.

Conservative treatment in chronic sinus disease is largely confined to alleviation of acute flare-ups. Sometimes it is necessary to resort to such minor operative measures as submucous resection of the nasal septum, turbinectomy and antrotomy, and the removal of diseased tonsils and adenoids, especially in children. The correction of allergic conditions I consider a very important part of conservative treatment.

In the relief of acute exacerbations, approximately the same procedures will suffice as those mentioned above under the treatment of primary acute conditions.

As was pointed out by Dr. Mithoefer, there are varying degrees of involvement in chronic sinus disease, and after careful diagnosis it may be necessary only to correct mechanical obstruction to secure relief, or operate on the key or activating sinus.

I have had little experience with diathermy as a curative agent and have little faith in its efficacy.

I recently heard a roentgenologist read a paper in which he claimed results so surprising to me that I mention them here. With

minute doses of from 20 to 30 r., chronic and acute sinuses, mastoiditis, and even cases of bronchiectasis were cured or relieved. This experience is not shared by several eminent roentgenologists of my acquaintance who feel that x-ray treatment is of no value. This I am prone to believe, because I cannot see any logical reason why a sinus filled with thickened infected membrane and polypi with encysted abscesses could be benefited by x-rays.

Antrum puncture in acute cases is inadequate and dangerous, as the antrum is frequently filled with edematous membrane, and the puncture is adding insult to injury and occasionally results in a fatality. This procedure in chronic cases as a diagnostic measure is rarely necessary if good x-rays are available. As a curative measure, except perhaps in subacute conditions, it is worthless.

The allergist can be of the greatest assistance to us in dealing with chronic sinus disease, and all allergic cases should be investigated before any radical type of treatment is instituted. Dr. Hansel[2] and Dr. Mithoefer both stress the importance of careful study of the discharge from these sinuses, both from a diagnostic standpoint and as an index to the kind of treatment most likely to be effective. I thoroughly approve of this and think it is too often neglected. Many allergic subjects become secondarily infected due to blockage of drainage and require a combination of allergic and operative rhinologic treatment.

In the surgery of chronic sinusitis, the ethmoid, because of its anatomical relation to the other sinuses, is most important. The surgical approach to it depends on the individual training and judgment of the operator and on his ability to make a correct diagnosis. Those of us who were Mosher trained can safely use the intranasal route. In dealing with pansinusitis, when the antrum and ethmoid are primarily involved with slight involvement of the frontal, I use the intranasal route in dealing with the ethmoid and do a radical antrum of the Caldwell-Luc type. If, on the other hand, the frontal is badly infected, with the symptoms during an acute exacerbation largely frontal in character, I use the external route and use a modification of my own of the non-obliterative Killian type[3] or a Lynch operation. In an extremely large frontal I often find it advisable to do an obliterating type of operation in the beginning. In making my decision as to which method to follow, I take into consideration the occupation, resources, intelligence, etc., of the patient, all of which are important for a satisfactory result. I discussed these factors at length in a paper given in Philadelphia last November.[4] In many cases the posterior wall is involved and has

to be removed. When this is done, complete obliteration is effective and healing prompt. Dr. Mosher has for some time advocated this as a routine in obliterating operations. I have found that most failures in frontal sinus surgery arise from neglect to remove the floor and orbital prolongations. If infected mucous membrane extensions are left in these or pockets where this can reform, failure is certain.

The after-care in postoperative sinus disease is of the utmost importance and perhaps as many failures are due to this neglect as to poor surgery. I think it would be impossible to put too much stress on the follow-up of postoperative sinus conditions. This is particularly true in allergic cases. Repeated allergic attacks tend to block drainage and predispose to reinfection.

One of the most controversial points in allergy and rhinology is the efficacy of sinus surgery in asthma. Whether the asthma associated with chronic sinus disease is due to secondary bacterial sensitization has not been proved. Nor does it matter greatly. The important thing is for the surgery to be thorough and the after-care adequate. My results compare favorably with those reported by Cooke and Grove.[5] All of my cases had been unsuccessfully treated for periods ranging from one to ten years by the allergist before coming under my care. Most of them required a follow-up of from one to two years after operation. I believe that those who fail to get similar results are not thorough enough in their surgery or neglect to follow their after-treatment closely. At the risk of being accused of prejudice, I think we, as rhinologists, can do more for these patients by ourselves than the allergist can by himself. The ideal combination is teamwork between the two, with the rhinologist in command.

In acute strepto- and pneumococcic infections of the sinuses, and even when the meninges are secondarily involved, we now have chemical agents which frequently give immediate results. We have had several cases on service recently at the Massachusetts Eye and Ear Infirmary of acute sinus disease and meningitis which recovered after the use of sulfanilamide, and I have a number of private cases of acute sinus infection which did not yield to ordinary measures but which cleared up under sulfanilamide treatment without operation. I do not believe in giving the drug just because one has a culture showing a hemolytic streptococcus infection, but prefer to reserve it for emergencies after the patient has demonstrated that he cannot successfully cope with the infection himself. When using this drug, I give it in large initial doses, getting the blood level up

to fifteen to twenty milligrams per cent in twenty-four to forty-eight hours, and keep it there for a considerable period of time. When the patient shows signs of recovery and has a negative culture I gradually reduce the dosage over a seven to ten day period. Transfusions of the immune variety are frequently used in conjunction with sulfanilamide and sulfpyradine therapy with good results. Transfusions of the non-specific variety are often beneficial in supplying a certain amount of antibodies which enhances the action of the drug.

In considering the problem of the existing prejudice against surgical treatment, we must admit that part of the blame is ours. There are many men practising our specialty throughout the country who are not competent to do major sinus surgery. Their failures reflect on all of us. This Association and others are doing much to improve this condition, but before this prejudice can be entirely removed we must first educate the general practitioner and through him the public. As an example of lack of knowledge among members of the profession at large, I recently attended a meeting of the Credentials Committee of a national society, and to my surprise, found many of those present had never heard of the American Board of Ophthalmology or Otolaryngology.

The average practitioner seems to be distrustful of our specialty in particular. He is loath to refer the patient until some serious complication has arisen. As a result of this, if the case becomes a protracted one or terminates fatally, the specialist is obliged to take the responsibility. It is not surprising, then, that the public has become skeptical of the ability of the rhinologist to operate and get a good result. It is becoming more and more difficult to convince patients that sinus surgery can be successful. I frequently hear the statement, "Once a sinus, always a sinus" and "one operation always leads to others."

520 Commwealth Ave.

BIBLIOGRAPHY

1. Mithoefer, William: Pertinent Facts Relating to Chronic Pansinusitis. Annals of Otology, Rhinology and Laryngology, 48:158 (Mar.), 1939.

2. Hansel, French K.: Allergy in Otolaryngology and Its Relation to Other Manifestations. I. General Considerations. Annals of Otology, Rhinology and Laryngology, 48:54 (Mar.), 1939.

3. Porter, Charles T.: Surgery of the Frontal Sinuses: A New Modified Killian. Transactions of the 29th Annual Meeting of the Amer. Laryng., Rhin. and Otol. Soc., Inc., p. 77, 1923.

4. Porter Charles T.: Some Observations of the Present Status of Frontal Sinus Surgery. Read at the meeting of the Section on Otolaryngology of the College of Physicians of Philadelphia, (Nov. 16), 1938.

5. Cooke, R. A., and Grove, R. C.: Relation of Asthma to Sinusitis with Special Reference to the Results from Surgical Treatment. Arch. Int. Med., 56:779, 1935.

Clinical Notes

LXII

NASAL TEETH

REPORT OF A CASE*

L. R. MARSHALL, M.D.

BATAVIA, N. Y.

Nasal teeth are not frequently encountered. They may give no hint of their presence or else give rise to a number of varied signs and symptoms. Occasionally they are found during the course of a routine examination of roentgen-ray plates for some other condition such as sinusitis. Hitschler has recently reported a case together with a discussion and a bibliography. As he states, there are few available references on the subject in the English language. Free use has been made both of his report and bibliography and acknowledgment is hereby made of that fact.

A survey of the literature made by Kramer in 1932, revealed that one hundred cases had been reported. Few cases have been reported since, hence the reason for this presentation.

The type of teeth found in order of their frequency are maxillary, first incisors, canines, premolars and molars. Deciduous teeth are rarely found in the nose. About one-half of the teeth are supernumerary, and as a rule only one tooth is found in the nose.

The etiologic basis for nasal teeth is not definite. They are found most frequently in individuals with supernumerary teeth. Developmental anomalies and structural defects favoring displacement of the dental anlage or obstructing the eruption of teeth are probably important contributory factors.

The symptoms listed by Hitschler are: (1) unilateral obstruction to breathing; (2) chronic nasal discharge, usually foul-smelling; (3) crusting; (4) localized ulceration. Other symptoms met with

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less frequently, according to this author, are epistaxis, conjunctivitis, fistula into the mouth, headache and facial pains resembling tic doloreaux. Reflex symptoms such as asthma, otalgia and laryngospasm have been reported. In some cases the tooth may be seen in the nose during an examination. It may, however, be covered by mucous membrane through which it sometimes ulcerates. When palpated, it is found to be of ivory hardness and slightly movable. Nasal teeth must be differentiated from exostoses, sequestra, dental tumors and cysts. Both lateral and occlusal x-ray studies should be made.

The treatment is surgical removal.

REPORT OF A CASE

CASE 1.—C. O'D., a white male, age 45, was admitted to the hospital of the U. S. Veterans' Administration on August 21, 1937, with a complaint of severe epistaxis for nine days. The medical history was essentially negative except that he had been treated for sinusitis on the left side for sixteen years. Nine days prior to admis-

sion he began to bleed from the left side of the nose, and in spite of tight packing he continued to lose considerable blood.

General physical examination was negative except for evidence of acute anemia. The patient, too weak to walk, was seated in a wheel chair and gave the appearance of illness.

Nasal examination showed the left nasal passage to be plugged with pledgets of cotton which were saturated with serum. Upon their removal, a brisk hemorrhage occurred. The nose was cleaned of clots by irrigation with a warm solution of one part hydrogen peroxide to three parts water. It was then packed with a weak solution of cocain and adrenalin, which checked the bleeding sufficiently to make a search for the bleeding point possible. It was determined that the bleeding point was located under the left inferior turbinate. A pledget of cotton was forced between the overhanging inferior turbinate and the naso-antral wall with complete control of the hemorrhage. Further examination indicated that both antra were dark on transillumination. The left middle turbinate was large and cystic. No free pus was noted in either the middle or superior meatuses.

The laboratory findings were as follows: Blood: red blood count, 3,110,000; white blood count, 9,000; hemoglobin, 65 per cent; Wassermann and Kahn, negative.

X-ray Report: Both maxillary sinuses are hazy, especially the left. What appears to be a fully developed canine tooth is seen projecting into the left inferior meatus of the nose. Its root is in contact with the lateral surface of the inferior turbinate bone, while its crown is buried in the floor of the nose in line with the naso-antral wall.

Dental Report: Gingivitis present. No caries. The following teeth are missing: Upper right: first and second molars, first bicuspid. Upper left: first cuspid. Lower left: first and second molars. Lower right: first molar.

The nasal tooth noted is not supernumerary, as can be seen from the above report. It represents an unerupted tooth. From an etiologic standpoint it is interesting to speculate whether or not a maxillary sinusitis on the left could have been present since childhood, thus influencing the normal position of the tooth.

The tooth could be easily palpated with a probe passed under the inferior turbinate. This maneuver provoked another attack of

epistaxis, indicating that ulceration of the mucosa of the inferior turbinate by the tooth was responsible for the hemorrhage which had brought the patient into the hospital.

After preliminary treatment for his acute anemia, the patient was prepared for surgery. Under local anesthesia obtained by topical application of cocain flakes and adrenalin 1-1000, the left inferior turbinate was infracted upwards. A number of futile attempts were made to grasp the tooth with forceps of various types. Its ivory, hard, smooth surface offered no secure hold. An attempt was then made to perforate the naso-antral wall posterior to the tooth with a Wiener rasp with the idea of loosening it. This method likewise failed, as it seemed too securely embedded. The patient was returned to the ward.

A number of days later, a modified type of Denker radical antral operation was performed on the left. The crown which was firmly embedded in the floor of the nose was exposed by the removal of the surrounding bone by mallet and gouge. It was necessary to remove the anterior third of the naso-antral wall in order to approach the tooth. With dental forceps the tooth was securely grasped and removed through the antrum. The antrum was lightly packed with a strip of vaseline gauze, the end of which was brought out into the nose through the antrostomy to facilitate removal. Closure was performed in the conventional manner.

Convalescence was uneventful, and when last heard from, one year later, the patient was free of all nasal symptoms.

VETERANS' ADMINISTRATION.

BIBLIOGRAPHY

1. Hitschler, Wm. J.: Nasal Teeth: Report of a Case. Arch. Otolaryng., 28:911 (Dec.), 1938.

2. Stafne, E. C.: Supernumerary Teeth. Dental Cosmos, 74:653, 1932.

3. Downs, W. G., Jr.: Studies in Causes of Dental Anomalies. Genetics, 12:570 (Nov.), 1927.

4. Abercrombie, P. H.: Eruption of Canine Tooth Into the Nasal Fossa Attended by Rhinitis Caseosa. J. Laryng., Otol., 40:586 (Sept.), 1907.

5. Wilms: Zahn im Nasenseptum. Deutsche Monatschr. F. Zahn., 25:725, 1907.

LXIII

LARYNGOPTOSIS

J. E. Mulsow, M.D.

Milwaukee

Chevalier Jackson[1] first described and named the condition known as laryngoptosis. The term is applied to an abnormally low position of the larynx. The larynx may be so low that the first ring of the trachea is level with the upper border of the sternum. In some cases the entire larynx may be down behind the sternum. Laryngoptosis of a slight degree is not uncommon, very marked cases are rare and few have been reported, C. Jackson and C. L. Jackson.[2] Tucker[3] reports a case in which ptosis of the larynx was due to downward displacement of the hyoid bone.

Etiology: Ptosis of the larynx (Jackson) may be of several types: (1) Congenital. (2) Ptosis due to developmental deficiency. (3) Secondary to pathologic changes, i. e., fibrotic contraction or a downward displacement of a growing tumor mass pulling downward on the trachea and larynx. (4) I believe we should include ptosis due to injury.

The diagnosis of laryngoptosis is made by palpation of the neck and noting the location of the larynx in its relation to the supra-sternal notch. The larynx may appear normal on indirect examination.

If the condition is simply one of low position and unassociated with any pathology, no treatment is indicated. There may be no symptoms except an abnormal tone of voice.

Report of a Case

Case 1.—On February 2, 1938, Mr. B., aged 52 years, was referred for examination of the larynx following an injury to his neck.

Previous History: The patient stated he was in an automobile accident in May, 1937, in which he sustained several fractured ribs and an injury to his neck. Shortly after the accident he had some

Laryngoptosis, showing larynx at rest.

difficulty in swallowing and he noticed a lump low down in his neck. The condition in his neck had never entirely cleared up, but it felt better until about four or five months after the accident. Then his throat again bothered him, he had coughing spells and difficulty in breathing. He said at times he was quite hoarse. At no time did he expectorate any blood.

Physical examination showed a well developed and well nourished man. He was of the short, husky type. He held his head in a perfectly normal position. There was no discomfort noted when the patient moved his head in various positions, i. e., forward, back-

ward or to either side. The tissues of the neck were soft and pliable on palpation, the thyroid cartilage was very low down and a portion of the cartilage was felt in the supra-sternal notch. The hyoid bone was also low and in close proximity to the thyroid cartilage, the anterior portion seemed to rest in the thyroid notch. The thyroid cartilage was tilted forward, downward and slightly to the left. There was no adenopathy noted. The patient had no difficulty in articulation nor in protruding the tongue beyond the lower teeth. Phonation was clear and the tone very deep or low. He had a monotonous voice. On several examinations no hoarseness was noted.

Examination of the nose and throat was essentially negative. A fairly good view of the larynx could be obtained with the mirror. The motility of the arytenoid cartilages and the vocal cords appeared to be normal. Direct examination was not made.

Roentgen examination (lateral films) of the soft tissues of the neck revealed the hyoid bone at about the level of the sixth cervical vertebra, constituting a downward shift of about two vertebræ from the normal position. The thyroid cartilage, which was partially ossified, was situated at about the level of the seventh vertebra and was tilted forward. Due to the ossification, together with the slight tilting to the left and forward of the thyroid cartilage, it was impossible to demonstrate the ventricles and other normal structures ordinarily seen in lateral roentgenograms of the soft tissues of the neck. There were no tumor masses seen in either the soft tissues of the neck or in the mediastinum. Antero-posterior studies showed the course of the trachea to be somewhat tortuous, its position being a little to the left of the midline at the level of the first thoracic segment and a little to the right of the midline at the level of the second. In the lateral chest film study, the position of the trachea and the size of the lumen appeared normal.

On fluoroscopic examination during the process of swallowing the usual motion of the hyoid bone and the larynx were noted and appeared to be quite normal.

COMMENT

There being no evidence of any pathologic condition associated with the low position of the larynx, no treatment was necessary. Had the condition been due to injury, one would expect to find some disturbed motility of the neck, difficulty in the protrusion of the tongue and expectoration of blood immediately following the

accident. There would unquestionably have been evidence of some impairment of the nerves or the arterio-venous and lymphatic systems supplying the soft tissues of the neck. The motor mechanism of the pharynx, larynx and the esophagus appeared to be quite normal.

The condition was probably a congenital one.

231 W. Wisconsin Ave.

REFERENCES

1. Jackson, Chevalier: Peroral Endoscopy and Laryngeal Surgery. Laryngoscope Co., St. Louis, 1915.

2. Jackson, Chevalier, and Jackson, Chevalier L.: The Larynx and Its Diseases. W. B. Saunders Co., Philadelphia and London, 1937.

3. Tucker, G.: Laryngoptosis: Ptosis of the Larynx Due to Downward Displacement of the Hyoid Bone Resulting from Fibro Stenosis and Shortening (Congenital Anomaly) of the Left Sternothyroid Muscles. Arch. Otolaryng., 25:389 (April), 1937.

LXIV

ALLERGY OF THE LARYNX

With Report of Two Cases

C. S. Chang, M.D.*

Peking, China

In 1882, Quinke first called attention to a definite entity characterized by a sudden appearance of localized and circumscribed edema in various parts of the body. He named this condition "acute circumscribed edema." Since his publication, the condition has been described under various other names: Quinke's disease, Quinke's edema, functional edema, angioneurotic edema, hereditary edema, and giant urticaria. Later, in 1888, Osler[1, 2, 3, 4] emphasized the association of the swellings with symptoms of gastrointestinal irritation, and further illustrated the striking influence of heredity. In his first communication he reported the disease occurring in five generations. Nineteen of the thirty-nine members of the family had angioneurotic edema and two died of edema of the larynx. Later, in three communications, he reported twenty-nine cases and considered the symptom as related to the erythema group of skin diseases.

As allergic manifestations may appear in any part of the body, the larynx is naturally not exempted from the affection. Among the various parts of the respiratory tract, the larynx constitutes one of the possible sites for allergic involvement.[7] Allergy of the larynx may be manifested in the form of angioneurotic edema or urticaria. The former is more common than the latter. The forms are frequently interchangeable.[7]

Allergy of the larynx may occur alone or in association with involvement of other parts of the respiratory tract. Wason[5] reported a fatal case of angioneurotic edema of the larynx and pharynx with involvement of the lungs. The condition may also be associated with other manifestations of allergy; for instance, skin manifestations, urticaria and eczema, headache, gastrointestinal symptoms, or various combinations.

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*Research Fellow in the Department of Otolaryngology, School of Medicine, Washington University, St. Louis, Missouri.

Allergic manifestations of the larynx are usually acute or temporary with very rapid onset, often without preliminary symptoms. Indeed, the symptoms are often so sudden and distressing that prompt diagnosis and adequate treatment should be given in order to avoid a fatal outcome. Chronic or persistent angioneurotic edema of larynx is rare.

Allergy of the larynx may be caused by hypersensitivity to foods, inhalants, or chemicals, like prolonged use of potassium iodide.[16] Freudenthal[14] reported a case of chronic urticaria of larynx caused by sensitivity to foods. Occasionally it may follow administration of vaccine or serum. It is also known that angioneurotic edema of the larynx may occur in highly nervous individuals. Osler[1] believed that in susceptible subjects it may be caused by autointoxication from some toxin in the intestinal tract, and in a certain percentage of cases, the larynx is the first part of the body attacked.

As described by Griffith[6] and Wason,[5] the main pathology of the condition is that of an extreme edema. The edema of the laryngeal mucosa is due to contraction of the blood vessels which is followed by secondary dilatation and stasis with serous exudation. It affects not only the mucous membrane, but also the deeper connective tissue, and even the substance of the muscles, preventing the widening of the glottic chink during respiration. Contrary to the usual conception, the tissue covering the true vocal cords is also involved.

The symptoms vary from simple hoarseness of voice and cough to asphyxia as a result of laryngeal obstruction. Pain on swallowing may be present. The laryngoscopic examination usually shows pallor and edema of the mucous membrane with serous exudation. When marked edema is present, the motility of the vocal cords may be impaired.

The treatment consists of local or symptomatic and specific treatments. For symptomatic relief, the following measures may be employed: (1) An ice coil around the neck. (2) Local application of cold adrenalin, using the 1/100 solution. This may be supplemented by hypodermic injection of 1/1000. (3) Catharsis to flush out the toxins in the intestinal tract. (4) Intubation or tracheotomy. The specific treatment aiming at radical permanent cure should be directed at elimination of the offending allergens.

The following two cases were taken from the records of the St. Louis Children's Hospital. The first patient had angioneurotic

edema of the larynx with gastrointestinal symptoms, and the second patient had an additional involvement of the tracheobronchial tree. In both instances the condition was proved definitely to be caused by allergy.

REPORT OF CASES

CASE 1.—M. E. J., a white male infant, aged three weeks, was admitted to the St. Louis Children's Hospital on August 8, 1932, on account of diarrhea of six days' duration and difficulty in breathing of two days' duration. The history related by the mother was as follows: Patient, para II, was born with normal delivery. Six days prior to admission he began to have diarrhea. The bowel movements came on after each breast feeding. The stools consisted of greenish curds with some mucus. The patient was seen by a pediatrician, who treated the diarrhea with various formulas of evaporated milk, lactic acid milk, etc. No improvement was obtained. On the day of admission he was given the first feeding of a formula of whole milk, barley water and protein milk at 1:00 p. m., and another feeding at 6:00 p. m. The patient was found to be very sleepy and drowsy afterwards. At 9:00 p. m. the mother noticed that the infant had difficulty in breathing, and became cyanotic. It was immediately rushed to the hospital.

The family history revealed that the mother had hay fever, and the brother was sensitive to cow's milk, with eczema during infancy.

Physical examination on admission showed that the patient was well developed and well nourished, but quite dehydrated, and slightly cyanotic. Respiration was labored with constant inspiratory stridor. The chest findings were essentially negative except for harsh breath sounds due to stridor. The white count was 20,500 with 65 per cent polymorphonuclears, 23 per cent lymphocytes, and no eosinophiles. It was repeated on August 15th and showed 18,000 with 58 per cent polymorphonuclears, 24 per cent lymphocytes and three per cent eosinophiles.

Within three hours after admission the patient had two attacks of respiratory difficulty with cyanosis and general spasticity.

Direct laryngoscopy was performed immediately after admission. The mucous membrane over the arytenoids was found to be moderately swollen and congested. The motility of the larynx and vocal cords was normal. Intubation was soon followed by tracheotomy. A culture of the laryngeal secretion was positive for pneumococcus. Smears of the secretion revealed no eosinophiles.

Upon admission to the hospital the patient was placed on a diet consisting of breast and evaporated milk. His postoperative course was uneventful, and his general condition showed gradual improvement. When decannulization was started, however, on August 11th, the patient again had marked difficulty in breathing, with attacks of cyanosis soon after the tracheotomy tube was corked. Similar attempts were repeated on several occasions, but all failed.

In view of the positive family history of allergy, and the sensitivity of patient's brother to cow's milk in infancy, the patient's formula was changed to Sobee on August 12th. Decannulization was again attempted on August 16th. After this the patient could breathe freely through the larynx, and the tracheotomy tube was finally removed on August 19th. The tracheotomy wound healed five days later. The patient was discharged in good condition on August 24th.

CASE 2.—R. B., a white baby boy, aged fifteen months was admitted to the St. Louis Children's Hospital on July 27, 1935, with a history of having had attacks of "asthma" for ten months. The present illness started in October, 1934, when the patient began to have attacks of wheezing. The first attack lasted for three weeks. The second occurred in December, 1934, and lasted for two months. The third occurred in March, 1935, and lasted for two weeks. The last attack started three weeks prior to admission. During each attack the patient had marked wheezing and attacks of coughing, with difficulty of breathing, day and night. Fever and vomiting were sometimes present. The parents did not know of any apparent cause for the attacks. The patient was fed on evaporated milk before the onset of the present illness. During the last two days before admission, the symptoms had become progressively worse. The respiratory stridor became more marked and the attacks of choking more frequent. The patient became increasingly restless, and was therefore brought to the hospital.

On admission physical examination showed that the patient was well developed and well nourished, but was in extreme respiratory distress and moderately cyanotic. There was marked inspiratory stridor with much retraction of the intercostal spaces. The voice was hoarse. The nose and throat were normal. The tonsils were enlarged but not inflamed. The chest findings were essentially negative except for distant breath sounds and a few rhonchi. The laboratory examination revealed a leucocytosis of 16,000 with 70 per cent polymorphonuclears, 25 per cent lymphocytes and no

eosinophiles. The Wassermann reaction was negative. The x-ray examination showed thymic enlargement.

An emergency tracheotomy was performed immediately after admission. During the course of the procedure, the patient ceased breathing but was quickly revived by supportive measures. Direct laryngoscopy revealed a moderate congestion of the epiglottis and vocal cords, and marked edema of the subglottic areas. The child was placed on a diet consisting of whole milk.

On August 2nd, laryngoscopy and bronchoscopy showed that the larynx remained edematous and more injected. The tracheobronchial tree was also edematous with much thin mucous exudation.

Decannulization was attempted on August 6th, but the patient became dyspneic every time the tracheotomy tube was corked.

On August 11th, a smear of the tracheal exudate revealed the presence of many eosinophiles. This suggested the possibility of allergy being the cause of the laryngeal edema. The patient's diet was changed on August 12th to a milk-free formula consisting of cemec, tomato juice and cod liver oil.

On August 13th, direct laryngoscopy showed a marked improvement in the laryngeal changes. The edema had disappeared and the mucous membrane had become normal. Decannulization, however, still could not be tolerated. On August 16th, the patient was placed on a whole milk diet again. Following this the laryngoscopy and bronchoscopy, repeated on several occasions, all revealed persistence of edema in the larynx and subglottic area with a large amount of mucous secretion. Decannulization, therefore, could not be accomplished. Various other therapeutic procedures, including adenoidectomy and x-ray treatment for the thymic enlargement, all produced no appreciable effect on the laryngeal condition. All skin tests, including milk, were negative.

On October 8th, the diet was again changed, with substitution of milk with Sobee. Twenty-four hours after institution of the elimination diet, there was progressive improvement, not only of the laryngeal condition but also in general health. The tracheal secretion became markedly diminished in amount. On October 12th, the tracheotomy tube could be closed without causing respiratory difficulty.

Tonsillectomy was done on October 21st because of marked hypertrophy. The postoperative course was uneventful.

Decannulization was started on October 25th, and was finally completed on October 28th. The patient was discharged well on November 13th.

A few days before dismissal whole milk was again given. Soon after he got home there were recurrent attacks of wheezing and coughing. The mother noted that the symptoms became improved after the diet was changed back to Sobee. On November 21st, the wheezing became worse with one attack of cyanosis. The nasal discharge and fever were also present. He was finally readmitted to the hospital. Later he was found to have acute nasopharyngitis. The symptoms and signs rapidly disappeared with the institution of a strict elimination diet and supplementary measures. The patient was discharged in good condition on November 24th.

SUMMARY

Two cases of laryngeal obstruction are reported, because they are particularly interesting and instructive from the following standpoint. In the first patient the pathological changes involved the larynx as well as the gastrointestinal tract. In the second patient, the tracheobronchial tree as well as the larynx was involved. In both instances, the disease was proved to be definitely allergic in origin with specific sensitivity to milk. Immediate relief was obtained by elimination diet. Allergy of the larynx no doubt occurs more commonly than is generally believed. In the management of all cases of laryngeal obstruction, the possibility of allergy as an etiological factor should always be taken into consideration.

REFERENCES

1. Osler, W.: Hereditary Angioneurotic Edema. International J. M. Sci., 95:362, 1888.

2. Osler, W.: On the Visceral Complications and Erythema Exudativum Multiforme. Am. J. Med. Sci., 110:628, 1895.

3. Osler, W.: On the Visceral Manifestations of the Erythema Group of Skin Diseases. Am. J. Med. Sci., 127:1, 1904.

4. Osler, W.: On the Surgical Importance of the Visceral Crises on the Erythema Group of Skin Diseases. Am. J. Med. Sci., 127:751, 1904.

5. Wason, I. M.: Angioneurotic Edema: Report of a Case with Necropsy Findings. J. A. M. A., 86:1332, 1926.

6. Griffith, T. W.: Remarks on a Case of Hereditary Localized Edema Proving Fatal by Laryngeal Obstruction. Brit. Med. J., 1:1470, 1902.

7. Hansel, F. K.: Allergy of the Nose and Paranasal Sinuses. The C. V. Mosby Co., St. Louis, Mo., 1936.

8. Halsted, T. H.: Angioneurotic Edema Involving the Upper Respiratory Tract. Tr. Am. Laryn. Assoc., 27:7, 1905.

9. Ballenger, W. L.: Angioneurotic Edema of the Larynx. J. Laryn. & Rhin., 10:266, 1896.

10. Källmark, F.: Case of Angioneurotic Edema of Larynx. Hygiea., 86:801 (Nov.), 1924.

11. Kennedy, A. R.: Angioneurotic Edema with Sudden Onset of Asphyxia. Brit. Med. J., 2:810 (Oct.), 1936.

12. Woodbury, F.: Urticaria Involving the Larynx and Causing Asphyxia. J. Laryngology, 13:522, 1898.

13. Taylor, J. M.: A Case of Urticaria of the Pharynx Producing Grave Edema of the Glottis. Philadelphia M. J., 1:607, 1898.

14. Freudenthal, W.: A Case of Chronic Urticaria of the Larynx. New York Med. J., 963 (Dec.), 1898.

15. Cohen, S.: Urticaria of the Larynx. Laryngoscope, 35:309, 1925.

16. Simpson, G. A. G.: Edema of Larynx Produced by Potassium Iodide. Brit. Med. J., 2:104 (July), 1924.

17. Theisen, C. G.: Acute Laryngeal Edema. ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY, 33:487, 1924.

18. Lyon, G. M.: Allergy in an Infant of Three Weeks. Am. J. Dis. Child., 36:1012, 1928.

19. Austrian, C. R.: Angioneurotic Edema. South. Med. J., 12:348, 1919.

20. Phillips, J. M.: Angioneurotic Edema. J. A. M. A., 78:497, 1922.

21. Brown, G. T.: The Treatments of Urticaria and Angioneurotic Edema. Ann. Int. Med., 3:591, 1929.

LXV

ACCIDENTAL INJURY TO THE LATERAL SINUS WITH REPORT OF TWO CASES

EDWIN C. BACH, M.D.

MILWAUKEE

Possible injury to the lateral sinus during the performance of a mastoidectomy, is probably the foremost thought in the mind of the otologist while operating. For this reason extraordinary caution is observed in removing bone from the area of the lateral sinus. However, the anatomical vagaries of the sinus, the use of poor or improper instruments, the occasional necessity for haste, and the blundering of an inexperienced assistant are some of the factors which make injury to the lateral sinus a reality, sooner or later, during the lifetime of the majority of otologists.

In the transactions of the American Laryngological, Rhinological and Otological Society of 1934, Henry Dintenfass presented a comprehensive paper covering the subject of accidental injury to the lateral sinus. He states that those whose patients have been fortunate enough to escape complications following this accident, do not consider injury to the sinus of serious import, except that it necessitates interruption of the operative procedure. That this mishap may not be entirely harmless, and that in a certain percentage of cases subsequent septic infection with ensuing complications, or even fatality occurs, was revealed in his investigation.

Septic thrombophlebitis and its complications constitute the chief danger from injury to the sinus. The incidence of this complication varies from 26 per cent to 43 per cent. Symptoms usually appear from four to five days following injury to the sinus. They may be evident within twenty-four hours or not until the twentieth day. As in spontaneous thrombophlebitis, there are chills, septic type of fever, blood changes, and later metastasis of septic emboli. Metastasis to the lungs, joints and subcutaneous tissues occurred in 15 per cent of cases reported in the literature.

Air embolism due to sinus injury, while a very infrequent complication, has occurred. It has been observed from two days to two weeks after the sinus has been injured, and usually occurs when the wound is dressed and the packing removed. The symptoms are

preceded by a hissing or sucking sound in the sinus area. This is immediately followed by shock, cyanosis and faintness or unconsciousness. The symptoms usually disappear promptly.

Precautions which might be observed to prevent sinus injury are as follows:

Anatomical variations of the sinus may be disclosed by x-ray examination, and the operation may be modified, if necessary, to avoid the lateral sinus. If the sinus lies close to the posterior canal wall, an incision as for a radical operation may be made and the mastoid antrum approached anteriorly by taking down the canal wall.

When a peri-sinus abscess is present, the sinus wall has been known to become considerably weakened, and has been ruptured by careless sponging, or by scraping granulations with a curette.

When a second mastoid operation is necessary, the sinus may have been exposed by the previous one, and is more superficial and adherent to the periosteum. Perforation at the time of the initial incision may occur.

The operative technique is a matter of some importance. The usual instruments for bone removal are the gouge, curet and rongeur. According to Dintenfass, the curet was the chief offender in producing injury to the lateral sinus. The injury was usually caused by using a small curet and trying to remove an excessive amount of bone with a single stroke. The gouge, especially of a large size, is the safest instrument for the removal of bone. The rongeur causes trouble when used to break bone rather than cut it. When a bite is made it should be cut through and care must be taken not to lift on the instrument and produce a prying effect, because splinters result and they may penetrate the sinus. Probes, retractors, and the careless use of the knife in making the radical flap have caused sinus injuries.

Treatment: The accepted method of treatment after sinus injury is to check hemorrhage immediately by pressure with a sponge, expose the sinus above and below the injury, and pack between the sinus and the bone with a gauze drain sufficiently to obliterate the sinus lumen. The operation may then be completed and the mastoid packed in the usual way with the wound left quite open. The dressings are changed as usual, but the sinus packs removed a bit at a time until about the eighth day, when all is removed.

If symptoms of sepsis supervene, ligation of the jugular and excision of the external wall of the sinus, with removal of clots, is indicated.

Sulfanilamide may be used to great advantage postoperatively in these cases.

REPORT OF CASES

CASE 1.—Mrs. A. K., age 47, was first seen at home on January 15, 1929, complaining of severe right-sided earache. She had been confined to bed for a week with influenza. A myringtomy was done and profuse drainage followed. The pain, however, persisted and involved also the mastoid area. On January 23 she was sent to the hospital, where an x-ray was made. The radiologist reported: Both mastoids are mixed pneumatic type. The left is clear; the right shows diffuse haziness and some areas of cell necrosis. The blood showed 17,600 leucocytes, 79 per cent being polymorphs.

Because of the patient's plea for relief of her pain, the x-ray report, and rather high leucocyte count, it was decided to open the mastoid on the following day.

While using the gouge over the sinus area, a small splinter of bone turned inward and punctured the sinus. Bleeding was insignificant and did not hamper completion of the operation. The wound was packed and partially closed in the usual way.

Culture of pus from the mastoid revealed a nonhemolytic streptococcus.

On January 26th she had a chill followed by a fever of 105 degrees. On January 27th an erysipelas appeared.

While removing the packing on January 27th, with the patient in a semi-recumbent position, a slight clicking sound was noted in the wound. The patient suddenly became dyspneic, went into profound shock and unconsciousness, and became cyanotic. She was immediately put flat on the bed, and on putting the ear to the chest, air could be heard churning in the heart. In about fifteen minutes she recovered consciousness, with no complaint except a slight headache.

The erysipelas subsided in six days, but her temperature did not stay normal long. From February 3rd to February 16th she ran an irregular fever which on some days reached 102.5 degrees. On February 16th the fever became more continuous, not going

below 100 nor above 102.5 degrees. On February 22nd the temperature reached 104 degrees and she complained of nausea and difficulty in swallowing. Examination of the larynx and hypopharynx revealed restricted movement of the right arytenoid. The soft palate appeared to move to the left and the tongue protruded definitely to the right. The fundi of the eyes were normal. A diagnosis of lateral sinus thrombosis, with involvement of the jugular bulb, was made. On February 25th, thirty-two days after the first operation, the jugular was tied and the mastoid reopened. Extensive thrombosis of the lateral sinus was present.

After a rather prolonged convalescence, she recovered.

CASE 2.—A. L., a female 13 years of age, gave a history of having had a left mastoidectomy in 1931, and another on the same side in 1932. She has been very susceptible to colds ever since and has had several postauricular abscesses with these colds. The ear was dry between these attacks.

On about June 1, 1938, earache on the left side appeared again. On June 27th the ear and old mastoid wound began to drain again. She ran a continuous fever which went as high as 103.5 degrees, and had pain radiating to the left side of the head, including the jaw.

On July 11th, she was admitted to the hospital, complaining of headache. Examination revealed a fever of 102.4 degrees and a pulse of 100. From a small fistula in the old mastoid wound and from the ear there was a moderate mucopurulent discharge. The right ear was normal. The patellar and ankle reflexes were increased. There was moderate stiffness of the neck. A positive Brudzinski sign and sustained ankle clonus were present. Spinal puncture revealed a turbid spinal fluid with 2,500 cells, 70 per cent polymorphs. No organisms present on smear or culture.

On July 12th she was prepared for operation, and on incising the skin just posterior to the old scar, the lateral sinus, which was just below the skin, was incised. Profuse hemorrhage was controlled by pressure and the sinus exposed above and below the injury. Packing with an iodoform gauze drain between the sinus and the bone controlled bleeding completely, and the operation was completed. The dura of the middle fossa was widely exposed and elevated over the tegmen. The wound was loosely packed with vaseline gauze and loosely sutured. She was given 500 cc. of saline intravenously and ten grains of sulfanilamide every three hours.

Her fever and symptoms subsided by lysis to normal in three days, and the sulfanilamide was reduced to forty grains daily. The last of the sinus packing was removed on the ninth day and she was discharged from the hospital on the eleventh postoperative day.

COMMENT

Case 1 illustrates, first of all, the grief which may befall early operation in acute mastoiditis. This operation was done on the ninth day after onset of otitis.

To avoid the complication of air embolism, it is important to do all mastoid dressings with the patient in a horizontal position, if the sinus has been opened at operation, even though the opening consists of a small puncture. This should be done for at least two weeks, postoperatively.

Septic thrombophlebitis may appear as late as three weeks after injury to the lateral sinus.

Case 2 illustrates the value of sulfanilamide when used postoperatively in complicated cases.

324 East Wisconsin Ave.

The Scientific Papers of the American Bronchoscopic Society

LXVI

PRESIDENT'S ADDRESS*

JOHN KERNAN, M.D.

NEW YORK

First I should like to thank the members of the American Bronchoscopic Society for the great honor conferred on me when they elected me president of this Society. As I was not present at the last meeting, I did not have an opportunity to do so at that time.

As a rule, papers read at a meeting such as this, and the presidential addresses, record work accomplished; difficulties overcome. I am going to discuss unconquered fields, the cases and circumstances in which bronchoscopy fails. We all know what bronchoscopy can accomplish, has accomplished in the past, and these accomplishments are actively being placed before the general profession. In order to grow, to advance, however, bronchoscopy must in the future succeed where in the past it has failed. Technique and results must be improved in old established uses, and new uses for the bronchoscope must be found.

Allow me to give an example. In one of the early editions of Dr. Jackson's book there is reproduced an x-ray of a chest which shows a straight pin far out in the periphery of the lung. In the text, that was designated as an irremovable foreign body, and yet many years later Dr. Jackson removed that very pin. The whole history of the use of the bronchoscope in foreign body work illustrates the point. The first bronchoscopes were crude and the forceps inefficient. Many foreign bodies were thought to be irremovable. Now the field of foreign body removal has been mastered. The

*Presented before the annual meeting of the American Bronchoscopic Society, Rye, N. Y., May 26, 1939.

tubes have been so perfected and the technique so well taught that many men all over the country now accomplish work that formerly could be done by very few.

Foreign bodies are no longer a problem. We cannot imagine meeting a question here that has not been solved in the huge experience of the Jackson clinic.

We must remember, however, that foreign body work is only a small part of bronchoscopy. The use of the bronchoscope has been extended to every lung condition. Any unanswered diagnostic or therapeutic problem in thoracic disease calls for a bronchoscopy. Can we attain the mastery in these other fields that has been attained in connection with foreign bodies?

This will call for a discussion not of the triumphs but the failures of bronchoscopy. (I want to say at once that these are probably personal failures.) To many of you they may not be failures at all.

We are asked many times to complete a diagnosis of malignant tumor of the lung by getting a specimen. The diagnosis may be substantially made, but for purposes of estimating possibilities of operation, x-ray treatment, prognosis, a biopsy is desirable. In all too many instances we introduce our bronchoscope and see no neoplasm. Let me say at once the appearance may be absolutely diagnostic to the trained eye of the bronchoscopist. There may be fixation of the tracheobronchial tree, loss of respiratory motion, even distortion and compression of the bronchial wall. At times there will be a tumor in the bronchial wall, over it intact mucous membrane. In these circumstances no specimen can be taken and the bronchoscopy, in the eyes of the medical man or surgeon, is a failure. Can anything be done to remedy this situation? It has seemed to me that sufficient use has not been made of the possibilities of finding clumps of tumor cells in the collected sputum. Using a flexible tipped cannula, many bronchi which cannot be seen into can be deeply aspirated. If a little saline solution is injected, carefullly collected and centrifuged, it is at times possible to find diagnostic clumps of cells.

This method of hunting for clumps of cells can also be used when the mucous membrane is broken by a neoplasm but there is no tumor formation offering a bite for the forceps. I suggest, under these circumstances, a small curette passed through the bronchoscope will dislodge cells which can then be collected by aspiration.

Another difficulty is that at times we can't see far enough. The lesion may be far down a small bronchus, or around a corner. If there were some method of projecting the light farther beyond the end of the tube, that might help. I have thought that proximal lighting would have that advantage, that it throws light deeper into the bronchial tree. Another gadget that might help would be a telescope on the proximal end of the tube, like an electric otoscope. Seeing around a corner may be accomplished in two ways. At times it may be justifiable to create an artificial pneumothorax. This straightens out the upper lobe bronchi so there is no longer a corner. There is also a long bronchial periscope which may be used to inspect the upper lobe bronchi.

We fail, far too often, alas, in explaining hemoptysis. A very serious question arises in these cases. Should we bronchoscope them during or soon after a hemorrhage? A bronchoscopy between bleedings will sometimes disclose the cause, a tumor, an ulcer, tuberculosis, or whatever it may be. But often in the intervals of bleeding, nothing is seen. In those cases it seems to me a bronchoscopy during hemorrhage is allowable unless the conditions are absolutely forbidding. Even though the cause cannot be demonstrated, the source may be localized. So much for failures in diagnosis. Now permit me to say a word about failures in treatment.

The first bronchoscopies in lung abscesses were done chiefly to rule out foreign bodies. Then use in lung abscesses was extended until it came to be recognized that a bronchoscopy with aspiration was a valuable means of cure in such cases. At the present time it may be said that a bronchoscopy is part of the routine examination and treatment of cases of lung abscesses. Many cases, even old cases, can be cured. Still, there are many cases which repeated bronchoscopies will not cure. Here is an unconquered field. What more can we do for the lung abscesses which at present we do not cure? Will improved methods of irrigation cure them? That is, attempts to get our irrigating solutions more surely into the abscess cavity.

This may be done in several ways. We make use of different shrinking solutions, such as adrenalin in saline. At present this is injected more or less blindly. We insert the tip of our flexible tube into a discharging bronchus and inject the solution. We have no assurance that the solution goes into the abscess rather than into normal lung. Would it not be better to do more of this work under the fluoroscope and to make sure, if possible, that our irrigating tube actually entered the abscess cavity? Little use has been

made of different positions during bronchoscopy. For upper lobe abscesses use of the lateral positions might be advantageous, as the left lateral positon for left upper lobe abscess and the right lateral positon for right upper lobe abscesses.

Another change which might make our treatment of lung abscesses more effective would be the use of a variety of disinfecting solutions. It has been reported that sulfanilamide solution is effective in sinuses infected with streptococci. Why not the use of such a solution in streptococcic lung abscesses? For the exceedingly foul abscesses due to mouth organisms and spirilla, salvarsan might be used locally in the cavity as well as intravenously. Another drug which promises something is zinc peroxide in thin emulsion. This has been used by the author with good result in a case of foul bronchiectasis. (These remarks on the treatment of lung abscess would also apply to bronchiectasis.)

Another method which has not been tried in lung abscesses, as far as I know, is ionizaton. As you all know, a chronic suppurating ear may at times be dried up by filling it with such a solution as weak zinc chloride and passing through a galvanic current. Could the same thing be tried in a case of lung abscess?

We know that under the guidance of the fluoroscope we can pick out cases in which the abscess cavity can be filled with lipiodol. Could not a weak zinc chloride solution be substituted for the lipiodol and a galvanic current passed through? If this were done would it do any good? Obviously a field for animal experimentation is open here.

The use of the bronchoscope in tuberculosis brings up many problems. A few years ago tuberculosis was a contraindication. Now many sanataria have bronchoscopic departments and bronchoscope nearly all their cases. We are often asked for specimens of sputum where there seems to be little. A careful washing of the suspected bronchus with saline and the examination of the centrifuged specimen often demonstrates tubercle bacilli.

Many questions come up in connection with tuberculosis of the bronchi. Should we do biopsies on ulcers and tumors? We see many cases with positive sputum. When bronchoscoped we find ulcerations or tumors. Should a specimen be taken to prove the nature of the ulcer or tumor. The question will be brought up as to the danger of spreading the disease. Having made a diagnosis of tuberculosis of the bronchi, what should the treatment be? There are men of experience who maintain that no local treatment

should be employed, that the local lesion will improve as the general lung condition improves. If treatment is decided upon, what should it be? Should such drugs as silver nitrate be used? This no doubt effectually heals the ulcers. But it has seemed to many that it promotes scar tissue as well as healing. A more effectual treatment, in the author's hands, has seemed to be the use of a small mercury vapor lamp. This appears to promote healing with less scar formation. These questions of the treatment of tuberculosis of the bronchi must be decided in the future after more experience accumulates.

What to do with strictures after they have formed is an interesting question. There is no doubt that in respect to those which have pyogenic cavities beyond the stricture it seems necessary to keep the stricture open for purpose of drainage. I have in mind cases which require periodic dilatation to prevent attacks of fever. On the other hand, in cases without pyogenic infection, where an attempt is being made to close a large cavity, it may be better to allow a bronchus to close, or even to close it on purpose. Certainly it is not logical to suppress the function of a lung by pneumothorax and keep the bronchus open. Certain cases will improve after such a complete closure of a bronchus.

On the other hand, I have seen cases in which the narrowed bronchus had ball valve action and the cavities could not be closed until the bronchus was dilated. It will be the task of the bronchopist to distinguish these different types of cases.

How should strictures be kept open? Mechanical dilatation is difficult and causes dangerous trauma. It will be understood that any trauma in the presence of tubercle bacilli opens a portal of infection. A more satisfactory method of dilating a bronchus has seemed to the author to be the use of a copper bougie through which is passed a weak negative galvanic current.

In some cases of tuberculosis the lesion seen in the bronchus is a generalized swelling of the mucous membrane, dusky red in color. The surface is unbroken. The appearance is like that of the nose blocked by swollen turbinate bones. I have not found a satisfactory treatment for this type of lesion. Applications of silver nitrate are irritating and have no effect. The patients are very irritable, cough a great deal, and in one case which the author bronchoscoped, there was an immediate spread to the other lung following the bronchoscopy. That is one of the dangers to be faced in bronchoscoping cases of tuberculosis, the danger of spreading the disease. These

patients are likely to cough a good deal. Now if the bronchi are anesthetized to suppress the cough, infected sputum may invade hitherto healthy lung. That is one objection to performing biopsies. If there is bleeding the blood may act as a vehicle for tubercle bacilli.

The above remarks are made in respect to intrabronchial tuberculosis. The question now comes up as to whether anything can be done for those cases which have extrabronchial tuberculosis, for instance, in the lymph glands. Tuberculous glands not broken down should be left alone. That seems obvious, but what about those which have formed abscesses and are pressing on a bronchus to obstruct it? We know that occasionally such an abscess will rupture, discharge through a bronchus, and if the patient survives the immediate danger, will heal. Will it ever be considered justifiable to incise such an abscess through the bronchoscope and actually use nature's method of healing them? It would require considerable courage to incise the intact wall of a bronchus.

The same applies to calcified nodes. Many of you doubtless have seen cases in which calcified nodes are pressing on bronchi, almost occluding them. Perhaps some of you have removed such glands after they have eroded into a bronchus. Will it ever be considered justifiable actually to incise the wall of the bronchus and remove them? So much for tuberculosis of the bronchi.

In regards to benign neoplasms. There is no difficulty about pedunculated tumors. These may be snared off or removed by forceps, and do not recur. It is different in regard to papilloma. The one case the author has seen in an infant, eventually died. Should we attempt fulguration, coagulation or implantation of radon?

Numerous reports have been made concerning benign adenoma. Some of these cases have been cured by mere forceps removal. In others the growth has been successfully destroyed by cauterization, or electric coagulation. The prognosis on the part of the bronchoscopist, however, should be very guarded. It may be that only a comparatively small part of the growth is intrabronchial. This indeed can be destroyed, but the extrabronchial part continues to grow. I have this past year seen two cases in which the growth had only seemingly been destroyed, and reappeared, in one case after ten years. Unless we bronchoscopists can devise some means of completely destroying these growths, the only remedy is a lobectomy. A word of caution is necessary here. If in destroying an adenoma too much normal tissue is destroyed, an intractable stric-

ture of the bronchus will result, with dangerous suppuration beyond the stricture.

A word as to malignant tumors of the bronchi. Certainly the bronchoscopist can do little for advanced tumors. I have seen no benefit from either insertion of radon or electric coagulation. Some palliation has been reported from the insertion of radium. But it is only brief respite which can be offered through these means. The assistance which we can give in making early diagnosis is our most valuable offering in this field.

It is only a very brief and sketchy review I have given you of a bronchoscopist's difficulties and failures. How may they be avoided and changed to successes? It seems to me much more intensive animal experimentation must be done. Many problems will be solved in the laboratory, but not all. We must remember that ours are clinical problems. Most of the answers will be found by pooling our clinical experiences as we do at these yearly meetings.

120 East 75th St.

LXVII

A CONSIDERATION OF THE REFLEX ETIOLOGY OF CARDIOSPASM*

BEN L. BRYANT, M.D.

LOS ANGELES

A certain amount of temerity is required to use the term "cardiospasm" in the title of a paper, inasmuch as Chevalier Jackson has stated frequently that the stenosis which is characteristic of the condition under consideration is not at the cardia, nor is it always spasmodic. Of the various appellations which have been suggested as substitutes for "cardiospasm," among which are preventriculosis, ingluviosis, functional hiatal stenosis, hiatal esophagismus, phrenospasm, and diaphragmatic pinchcock stenosis, perhaps the most satisfactory is achalasia, used by Hurst at the suggestion of Sir Cooper Perry, and derived from the Greek word meaning relaxation, preceded by "a," implying absence of. For whatever the remote or immediate cause, whatever the exact neuromuscular action in a given case, and whatever the duration of symptoms, they are due to a failure of the lower portion of the esophagus to relax during the deglutitory cycle.

Although some authors insist that cardiospasm is an acceptable term because there can be felt a sphincteric grip in some cases, this is probably due to the action of the crura, muscular and tendinous in structure, of the under surface of the diaphragm, which surround the esophagus. Nevertheless, the term cardiospasm persists throughout the literature, perhaps because of the veneration due age, for it was the term first used by Miculicz in 1888 to describe the condition. It is interesting to note that more than two hundred years before that date, Thomas Willis had described in 1672 a patient with the typical symptoms of achalasia, including the fact that "he cast up raw (or crude) whatever he had taken in." For the patient, Willis made a rod-like dilator of whalebone, through the use of which he was able to take nourishment for the ensuing fifteen years.

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Great as has been the discussion and varied the opinions regarding the nomenclature, they by no means exceed the variation in the ideas which have been expressed as to the etiology. It is not within the scope of this paper to discuss all of these in detail nor to evaluate them. Undoubtedly there are some congenital cases, and undoubtedly there are some which are purely psychogenic in origin, in addition to those in which a psychoneurotic factor is added to an immediate demonstrable cause. Witness the gentleman reported by Winkelstein whose symptoms were aggravated by financial difficulties, and who was finally relieved not by any of the accepted therapeutic measures but only by his marriage with a wealthy woman.

There are doubtless cases, also, where "liver tunnel" obstruction and pressure from surrounding structures is an etiological factor, and others in which the condition is due to fibrosis of the terminal portion of the esophagus caused by infection from contiguous organs. There are many theories of the origin of this condition which are known to all of us, and the very fact that there is so much disagreement among competent observers as to the findings as well as to the etiology would lead to the conclusion that it is impossible and probably always will be impossible to group all cases of achalasia of the esophagus under one heading.

It has, however, come to be more and more the consensus of opinion that there is always an underlying neurogenic cause in the sense of an immediate interference with the reflex pathways governing deglutition. The nerve supply of the area under consideration is from the myenteric plexus (Auerbach's plexus), which is a network of nerve fibers, mostly unmyelinated, which lies between the inner circular and outer longitudinal muscular layers of the enteric canal and which extends from the upper portion of the esophagus to the terminal portion of the rectum. This plexus is uniformly distributed on all sides of the alimentary canal. It has been demonstrated that the function of the myenteric plexus is to co-ordinate the movements of the gastro-intestinal tract. It follows, therefore, that whatever can interrupt these impulses can indirecly cause the failure of the lower end of the esophagus to relax for the passage of food. At the nodal points of the network, ganglions occur which in the upper part of the esophagus are terminal cells of the vagus nerve, but in the last few centimeters are combined with ganglion cells from the sympathetic system. Lendrum in his recent work, and Rake in earlier studies, found degenerative changes in this plexus in cases of long-standing achalasia.

Experimental studies have proven that either vagal failure or sympathetic overstimulation produces the so-called cardiospasm, and this is supported by reports of negative results from a therapeutic standpoint by section of the vagus, and of repeated good results with relief of symptoms by cervico thoracic sympathetic ganglionectomy and by celiac sympathectomy (Craig, Moersch, Vinson and Knight, et al.).

It has been observed repeatedly that ano-rectal lesions produce pyloric spasm, but peculiarly enough, one finds no reports of cardiospasm from such lesions. Obviously it is possible, and one wonders whether the symptoms of heartburn and substernal pain occurring in some cases of hemorrhoids and anal fissures are in reality caused by transitory achalasias of the esophagus which have passed unnoticed by the physician. The literature on achalasia contains many references to diseases of the abdominal viscera as causative agents, but usually the reference is vague.

The role played by the rectum in the production of achalasia was brought forcibly to my attention thirteen months ago when I was asked to see a 70-year-old white man who had been unable to swallow food or liquids for forty-eight hours. His difficulty had commenced suddenly at the time of his evening meal, and in the interim he had had recurrent spasms of epigastric and substernal pain accompanied by retching. A fluoroscopic examination made with barium before he was brought to me showed that none passed into the stomach from the esophagus. When the esophagoscope was introduced, the esophagus was found to be almost entirely filled with undigested food and barium, and when this was removed the esophagus was observed to be normal in appearance throughout until the lower end was reached. Here a sphincter-like contraction was found which prevented the further passage of the tube. A No. 14 Jackson esophageal bougie was passed through this area, no force being required, immediately upon which the area opened and the esophagoscope could be passed into the stomach. I learned afterwards that when a preoperative enema for the rectal instillation of nembutal was given, a large fecal impaction was released. On the following day the patient was swallowing all foods normally and was free of discomfort. He was given advice as to his diet and laxatives, and a check one year later revealed that he had had no further difficulty.

It seems certain that the fecal impaction was the causative agent in this instance, and equally certain that without proper advice to prevent the recurrence of such an impaction, further attacks of his

esophageal difficulty could be expected. It must be remembered that through the vegetative and cerebrospinal nervous systems, the anorectal region is in communication with every organ in the body. The rectum, sigmoid, and descending colon are supplied by the pelvic nerves, which take their origin from the sacral cord and pass into the pelvic plexus, which is connected with the inferior mesenteric plexus through the hypogastric fibers and ends in Auerbach's plexus.

Another striking example of reflex etiology is afforded by the case of a 62-year-old housewife who was referred with a complaint of difficulty in swallowing of seven months' duration. She had been under treatment in a neighboring state without improvement, and for four months had swallowed nothing but liquids. She weighed only ninety pounds, having lost more than thirty pounds of weight, was emaciated and sallow in appearance. Her red cells numbered 3,805,000, with a hemoglobin value of 58 per cent. Her history and appearance strongly suggested malignancy. Mirror examination of the larynx and hypopharynx revealed nothing abnormal except generalized paling of the mucous membranes. The x-ray examination made fluoroscopically and with films resulted in the following findings:

"After a barium filled capsule was swallowed, it reached a point slightly below the larynx, where it was obstructed in its passage. A thick barium paste was then taken and was shown to be obstructed at the same level. From an x-ray standpoint the findings would indicate an obstructive lesion in the esophagus at the level of the larynx, which most probably is a malignancy."

A direct examination with the esophagoscope was made under local anesthesia, the findings being as follows: The larynx and trachea so far as visualized were normal in appearance. The esophagus was entered easily, but at the level of the aorta the lumen was much narrowed. There was, however, no granulated or ulcerated surface, but a gradual blanching of the tissue, with the lumen narrowing to a fistula-like opening scarcely larger than the lead in a pencil. This was several centimeters below the level of the crossing of the aorta. Still having in mind the probability of a malignancy with an annular stricture above it, and not wishing to dilate the region forcibly if this were the case, I injected lipiodol into the stricture by means of a syringe with a long injection tip. Subsequent x-ray showed the lipiodol in the stomach with no demonstrable pathology. A second esophagoscopy was performed, and I quote from the note made at that time: "The stricture previously described was visualized and the tip of the smallest esophageal bougie

was introduced with the idea of performing a gradual dilatation. It immediately and easily slipped through the stricture, which dilated without force sufficiently to permit the free passage of the largest dilator."

The patient was able to eat comfortably following this procedure, and six days later, for purposes of observation and another dilatation, if necessary, the esophagoscope was passed again. At this time the esophagus was found to be normal in appearance throughout from the pyriform sinus to the stomach. Subsequent physical examination revealed the following interesting points: The test meal showed free hydrochloric acid 42, total acidity 68, lactic acid negative, and occult blood positive. The barium meal passed normally through the esophagus, which showed no stricture or diverticuli. The stomach was of the small, fish-hook type and a small crater was seen on the lesser curvature about one inch from the pyloric-duodenal junction. The peristaltic waves were slow, as was the emptying time. No abnormality was seen in the duodenum or elsewhere in the intestinal tract. Repeated examinations of the stool revealed no occult blood. A diagnosis of a healed ulcer of the stomach was made and the patient was placed on a postulcer diet and given cerium, bismuth and magnesia, because the slow emptying time of the stomach was considered to predispose to the recurrence of the ulcer. The patient was seen again after six months. She had had no further difficulty whatsoever in swallowing and had regained practically all of her lost weight.

Here we see a constant and practically complete closure of the lower end of the esophagus existing for a period of months, and resulting from a lesion in the stomach. It was probably fortunate in her case, because, inasmuch as the ulcer had not been diagnosed and treated, it is well that nature, by this reflex action, forced the patient to remain on a milk diet sufficiently long for the ulcer to heal. It might be added, however, that nature almost over-did it, because the patient was starving to death at the time of the dilatation.

A third case of interest, to note in passing, is that of a 36-year-old ophthalmologist who, last December, immediately after a day of skiing, and while overheated, gulped down a cold soft drink. There developed a severe cramp deep beneath the sternum and, according to his statement, he could feel the esophagus clamp down. The pain decreased considerably in about ten minutes, but returned from time to time for the following week, during which time he was able to take only sips of food. At no time was there regurgi-

tation. For five weeks thereafter he experienced marked difficulty in swallowing and only then presented himself for fluoroscopic and x-ray examination. Although no esophageal achalasia was demonstrated, he was invariably conscious of the passage of the barium through the cardia upon swallowing, this sensation being checked by fluoroscopy. A spasm of the pyloris was demonstrable. He has been relieved of his symptoms by atropin medication, except when he is under a particular nervous strain or mentally worried, at which times he still, five months later, has a recurrence of symptoms. This, of course, demonstrates the psychoneurotic factor observed in some cases.

This discourse is not intended to place all cases of so-called cardiospasm on a reflex basis. The cases cited, however, at least serve to support a statement, which has been made by Jackson and other writers, that anything which unduly disturbs the reflex arc of the myenteric plexus may serve as an inciting cause of functional stenosis of the esophagus. It suggests further that in intractable cases which are not relieved by mechanical dilatation, a meticulously careful search for lesions elsewhere which might act as etiological factors be instituted before sympathectomy is resorted to, and further it suggests that even in cases easily relieved by ordinary therapeutic measures, reflex etiological lesions should be sought, so that, if found, their correction can help prevent a recurrence of the achalasia.

2007 Wilshire Boulevard.

BIBLIOGRAPHY

1. Craig, W. M., Moersch, H. J., and Vinson, P. P.: Treatment of Intractable Cardiospasm by Bilateral Cervico-thoracic Sympathetic Ganglionectomy: Report of Case. Proc. Staff Meeting Mayo Clinic, 9:749-753 (Dec. 12), 1934.

2. Hurst, A. F.: Treatment of Achalasia of the Cardia—So-Called Cardiospasm. Lancet, 1:618, 1927.

3. Jackson, Chevalier: Diaphragmatic Pinchcock in So-Called Cardiospasm. Laryngoscope, 32:139 (Feb.), 1922.

4. Jackson, C. L.: The Treatment of Cardiospasm with Notes on Diagnosis and Etiology. Arch. Phys. Therapy, 15:172, 1934.

5. Lendrum, F. C.: Anatomic Features of the Cardiac Orifice of the Stomach, with Special Reference to Cardiospasm. Arch. Int. Med., 59:474 (March), 1937.

6. Mosher, H. P.: Cardiospasm. Laryngoscope (May), 1922.

7. Rake, G.: Epithelioma of Esophagus in Association with Achalasia of Cardia. Lancet, 2:682 (Sept. 26), 1931.

8. Starling, H. J.: Achalasia of the Cardia Associated with Chronic Gastric Ulcer. Guy's Hospital Reports, 85:197-200, 1935.

9. Winkelstein, A.: Psychogenic Factors in Cardiospasm. Am. J. Surg., 12:135-138 (April), 1931.

LXVIII

CARDIOSPASM: A PNEUMATIC-MERCURY DILATOR*

GABRIEL TUCKER, M.D.

PHILADELPHIA

Cardiospasm, "so-called cardiospasm," functional hiatal stenosis, preventriculosis, and achalasia are terms applied to a condition of closure of the lower end of the esophagus in the region of the diaphragm with a greater or less degree of dilatation of the thoracic esophagus. There is much difference of opinion as to the exact etiology and the natural history of the disease. That the condition starts as a functional condition in some cases and later becomes definitely organic in nature is well established.

TREATMENT

The treatment of cardiospasm that has given the most favorable results is gentle dilatation or stretching of the hiatal esophagus repeated over a considerable period. As a preliminary to the stretching procedure, a careful x-ray study of the lower end of the esophagus and cardia followed by direct examination with an open-tube that is long enough to enter the stomach (Fig. 5-B) should be carried out in every case. The esophagoscopic examination will eliminate the presence of ulceration or other lesion that might contraindicate the use of a dilator. Through the open esophagoscope an endoscopic pneumatic dilator gives very good results (Fig. 5-D). In a number of cases repeated dilatation is required. The use of the mercury bougie of Hurst, or the pneumatic dilator of Mosher, or some other type is frequently used to carry out the follow-up treatment. After the hiatal esophagus has been dilated esophagoscopically, the Hurst mercury bougie usually passes with very little discomfort to the patient. To increase the effectiveness of this method of dilatation, I have combined the principles of the mercury bougie and the pneumatic dilator.

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*Presented before the Annual Meeting of the American Bronchoscopic Society, Rye, N. Y., May 26, 1939.

A MERCURY FILLED PNEUMATIC CARDIOSPASM DILATOR

The mercury-pneumatic dilator (Fig. 5-A) is a combination of the mercury bougie and a pneumatic bag for dilatation of the hiatal esophagus in so-called cardiospasm. The instrument combines the principles of the Hurst mercury bougie and the Mosher pneumatic dilator. It consists of a rubber tube with a round, closed tip, size 36 French, 75 cm. length, the lumen of the tube filled with mercury. There is a smaller rubber tube which is connected mercury tight inside the large rubber tube through a one-piece metal connection with a dilating bag on the outside of the large rubber tube. The dilating bag is composed of silk and rubber with barium stripes, like the Mosher bag. The dilating bag is 15 cm. in length and surrounds the larger tube beginning at a distance of 10 cm. from its distal end. The connections are such that the bag may be inflated from the proximal end of the dilator, so that when the bougie is swallowed, the bag is carried with it through the esophagus; and when it is properly placed in the hiatal esophagus, inflation of the bag with air will carry out any desired dilatation in addition to that which has already been accomplished by the passage of the bougie containing mercury. The pressure in the pneumatic bag is controlled by a manometer such as is used on all pneumatic cardiospasm dilators. This type manometer was devised by Mosher and is made with an illuminated dial so that it can be seen in the darkness of the fluoroscopic room. The mercury pneumatic bougie because of its weight is readily swallowed with very little discomfort to the patient. The graduated scale on the proximal end of the dilator allows a record to be made of the proper depth of insertion of the bougie under fluoroscopic guidance. When the bag is at the proper level, the scale is read at the point where the rubber tube comes in contact with the teeth, and this reading can be subsequently used for carrying out dilatations without fluoroscopic guidance.

When I first demonstrated this pneumatic-mercury dilator at a recent meeting on April 5, 1939, of the Philadelphia Chapter of the Pan-American Medical Association, I learned that Soulas, of Paris, had devised a similar dilator. On investigation I found that the Soulas bag has mercury only in the tip, otherwise it was quite similar. My dilator, however, was constructed without any knowledge of the Soulas or any other similar instrument, other than the Mosher dilator and the Hurst bougie.

REPORT OF CASES

The mercury filled pneumatic dilator has not been in use long enough to state definitely its value. However, there have been sev-

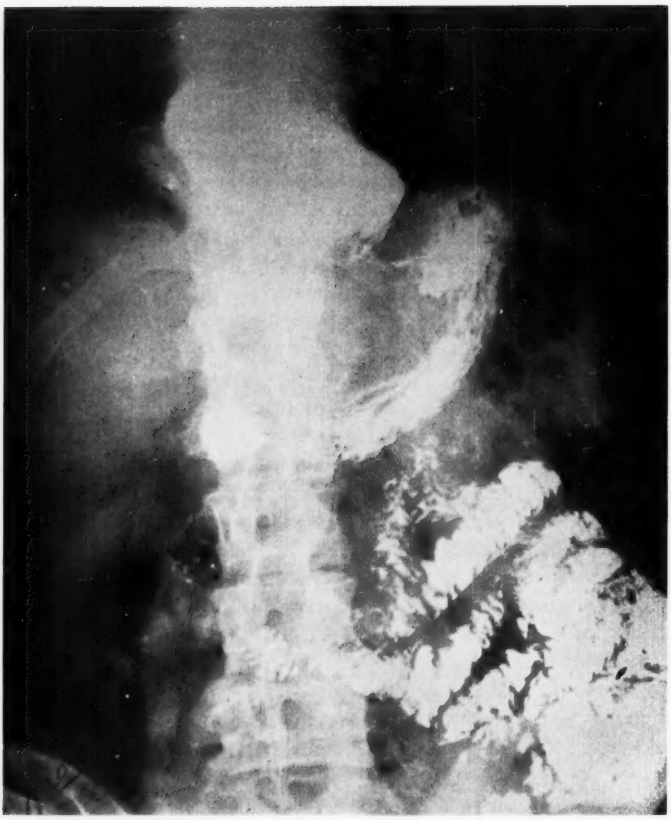

Fig. 1, Case 1. Film made in the upright position shows barium passing through the hiatal esophagus at right angles to the long axis of the esophagus. Film made by Dr. Eugene Pendergrass.

eral cases in which it seemed to have certain advantages, and their mention may be worthwhile.

CASE 1.—In the case of a man, 63 years of age, who had dysphagia for a period of twenty-eight years, we were able to enter the stomach by no other means. At the onset of the man's condition a definite diagnosis of cardiospasm had been made. Among other things, an Einhorn dilator had been used with a measure of relief obtained at first. Finally, the patient found that he was able to get food through into his stomach by taking sufficient food to fill completely the dilated esophagus, then by forcing down a half cupful of water and swallowing air while sitting in the upright position, he was able to get the contents of the dilated esophagus to pass suddenly through the hiatal esophagus into the stomach. The patient himself felt that he was completely emptying the esophagus, but on examination we found otherwise. For the past several years the patient had been accustomed every night at bed-time, after he had emptied his esophagus, as he supposed, to take from four to eight ounces of undiluted whiskey, following this with water. He stated that this passed readily into his stomach and after this he was able to sleep peacefully for the night. Examination showed a huge dilatation of the esophagus and the barium seemed to pass in a thin stream through the hiatal esophagus. This demonstrated that the opening through the hital esophagus was at right angles to the long axis of the dilated thoracic esophagus (Fig. 1. The film was obtained during efforts to empty his esophagus). There was marked fibrosis in this area and we were unable to get the esophagoscope through into the stomach even over a previously swallowed string. The patient was placed in the upright position in front of the fluoroscope, the esophagus emptied, and the mercury bougie passed under fluoroscopic guidance until it reached the beginning of the hiatal esophagus. Patient was then instructed to swallow air and press downward, and as he did so the mercury bougie slipped into the stomach. The mercury-pneumatic dilator was passed in the same manner and the hiatal esophagus dilated (Fig. 2).

Esophagoscopic examination showed no inflammatory reaction in the lower end of the esophagus. Mucous membrane seemed practically normal in color. This was surprising, because undoubtedly the man had had some retention even though he thought he was emptying his esophagus completely. It is suggested that his ingestion of four to eight ounces of straight whiskey may have had something to do with preventing inflammatory reaction in the dilated esophagus such as is generally seen in cases where there has been such marked retention.

Fig. 2, Case 1. Film shows the pneumatic-mercury dilator in the hiatal esophagus. The arrows indicate the point of constriction. Film made by Dr. Eugene Pendergrass and Dr. P. J. Hodes.

Fig. 3, Case 2. Pneumatic-mercury dilator in the hiatal esophagus. Its direction is normal. Arrows show constriction in bag position of narrowed hiatus. Film made by Dr. Eugene Pendergrass and Dr. P. J. Hodes.

Fig. 4. Barium passing through hiatus after dilatation. Patient was very much improved. Film made by Dr. Eugene Pendergrass and Dr. P. J. Hodes.

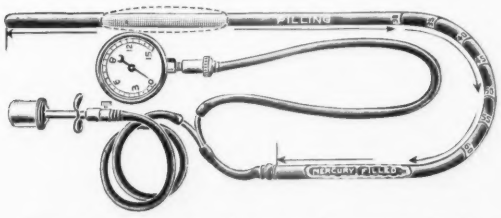

Fig. 5-A. The author's pneumatic mercury filled cardiospasm dilator.

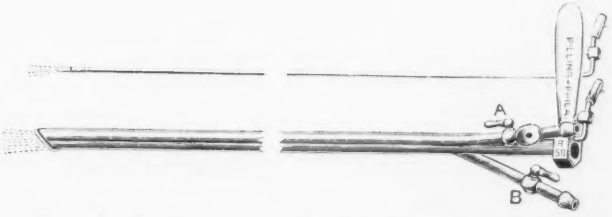

Fig. 5-B. Esophago-gastroscope 8 mm. x 58 cm. for examination and dilatation of the hiatal esophagus. In children a tube of similar construction 6 mm. x 53 cm. is used, and in new-born infants the author's infant esophagoscope 4 mm. x 35 cm. equipped with the Pilling special small lamp.

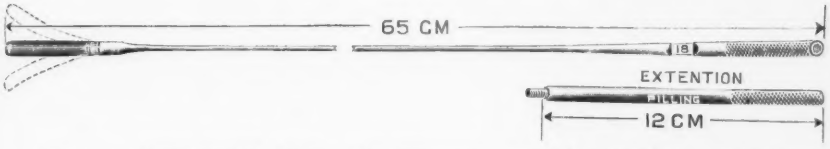

Fig. 5-C. Hiatal opening finder. This instrument is a modification of the Jackson lumen finder. Length is 65 cm. with an extension of 12 cm. The flexible tip is one-half the length of the Jackson finder and it is filled with barium so that it is opaque under fluoroscopic examination.

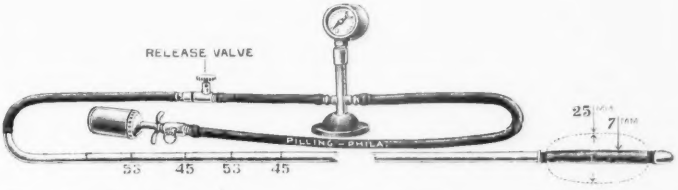

Fig. 5-D. The author's endoscopic pneumatic dilator presented at the San Francisco Meeting of the American Bronchoscopic Society, 1928.

CASE 2.—A young woman, twenty-two years of age, who had a cardiospasm from the the age of 16 years, had been treated by repeated dilatation, pneumatic, hydrostatic, and esophagoscopic, and and had had resection of the peri-arterial sympathetic with the hope that the hiatal opening would function properly, but only temporary improvement had resulted. The hiatal esophagus was very easily dilated with the pneumatic-mercury dilator (Fig. 3) and the results have been quite favorable up to the present time (Fig. 4).

COMMENT

The use of the mercury filled pneumatic dilator appears to be a safe method of stretching the hiatal esophagus in cases where this method of treatment is indicated. The instrument is easily introduced and produces no discomfort. The presence of ulceration in the hiatal esophagus should be excluded by direct examination in every case before stretching is used. It is believed that the mercury filled pneumatic dilator will prove a valuable addition to the instrumentarium for the diagnosis and treatment of "cardiospasm" (Fig. 5).

269 SOUTH NINETEENTH STREET.

LXIX

THE LATE RESULTS OF FOREIGN BODIES LONG RETAINED IN THE LOWER AIRWAYS*†

ETHAN FLAGG BUTLER, M.D.
N. STANLEY LINCOLN, M.D.
JOHN KENNETH DEEGAN, M.D.
RALPH HORTON, M.D.

ITHACA, N. Y.

INTRODUCTION

The purpose of this paper is to discuss the nature and significance of pulmonary pathology consequent upon foreign bodies that have been long retained in the airways. The discussion is based upon nine cases of proved foreign body which have been collected over a period of ten years. The nature of the pathology has been established by autopsy, operation or roentgenologic examination. Four cases occurred in a series of 766 thoracic surgical patients seen in northern Pennsylvania and Southern New York; a case incidence of 0.52 per cent. Four more have been discovered among 273 patients with nontuberculous pulmonary disease, admitted during 1936, 1937 and 1938, to the New York State District Tuberculosis Hospitals at Mount Morris, Ithaca and Oneonta; a case incidence, in this group, of 1.46 per cent. An additional case of many years ago has been contributed by Dr. Max Pinner, formerly pathologist of these hospitals, from his past autopsy records.

In eight of the nine cases the nature and the location of the foreign body is fully proved. In the remaining case the fact of the foreign body is established by the bronchoscopic recovery of vegetal foreign matter, the nature is predicated on the history, the location is predicated on bronchoscopic observations made within two years of aspiration.

Six patients were male, three were female. All were white. At the time of aspiration, five patients were three years of age or less,

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*From the New York State District Tuberculosis Hospitals.

†Read at the 22nd Annual Meeting of The American Bronchoscopic Society, Rye, N. Y., May 26, 1939.

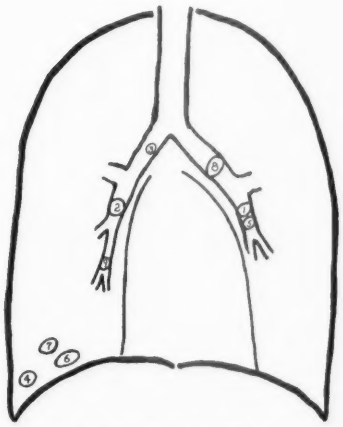
two in their second decade, one in her third decade and one of unknown age. At time of examination, three patients were in their first decade, four in the second decade, one in the third decade and one of unknown age.

Fig. 1 sets forth the location of the foreign bodies within the airways and indicates whether they caused significant bronchial obstruction. The subjoined table (I) catalogues their nature and duration of residence. The shortest recorded sojourn was two months; the longest was estimated at sixteen years. In two cases it was of unknown duration. Only once was the length of residence a matter of relatively little consequence; in all others, the prolonged residence, plus bronchial obstruction, caused pathology which either killed or crippled the victim. From the diagram it will be seen that six invaders lodged on the right, three on the left. Three passed rapidly to smaller bronchi and were found near the periphery of the lobe. Four foreign bodies were metallic, four were vegetal, one was rubber.

It is interesting to analyze the factors that led to delay in diagnosis and treatment. In five instances the family physician apparently overlooked or disregarded a clear-cut history of a foreign body accident. Of these five patients, two are dead. Twice there was delay in securing roentgenograms which would have revealed a metallic foreign body. Once serial roentgenograms were repeatedly misinterpreted. In another instance the foreign body eluded search by all methods until found in the excised lobe. One patient elected to ignore a promptly diagnosed foreign body.

Bronchoscopy was employed in eight cases, but only once was the foreign body successfully removed. Twice disintegrated fragments were recovered; in one case presumably the entire residue of foreign material, in the other case but a small portion of the mass. Bronchoscopic search failed, in four instances, to even visualize the invader.

Four of the nine patients died, a series mortality of 44.4 per cent. One died of metastatic brain abscess, a not unusual terminal event in the evolution of chronic pulmonary suppuration. Two died shortly after bronchoscopy performed during the height of acute suppurative pneumonitis. The fourth died after surgical lobectomy, urgently demanded by otherwise uncontrollable pulmonary hemorrhage.



Location, nature and length of residence of the nine foreign bodies. The numbers refer to the cases, as listed in "Case Reports." The obstructive effect of the foreign body is indicated by the relation of the encircling line to the bronchial wall.

TABLE I

1.	Wire nail	16 years
2.	Wire nail	10 weeks
3.	Open safety pin	3 years
4.	Sliver of wood	5 months
5.	Metal screw cap	? years
6.	Timothy head	1 year
7.	Timothy head	6½ months
8.	Gum rubber	2 months
9.	Bean	? weeks

REPORT OF CASES

CASE 1.—M. M., 19 years old, white school girl, was seen in April, 1929, because of cough, foul sputum and pulmonary hemorrhages. History, at that time, disclosed that when the patient was three years old she had had "whooping cough," which had been followed by persistent "bronchial trouble." In October, 1927, there had been two moderate pulmonary hemorrhages. In July, 1928, there was an attack of "pleurisy" and several slight hemorrhages, followed by productive cough and much foul sputum. Pulmonary tuberculosis was suspected, and she was treated for this disease, even

though the sputum was never examined. In November, 1928, a consultant reported clubbing of fingers, fetid breath, restricted motion in the left lower chest, other signs of pathology involving and apparently limited to the left lower lobe, sputum negative for tubercle bacilli, and fluoroscopic evidence of a metallic foreign body in a greatly shrunken left lower lobe. The foreign body was identified as a wire nail. Bronchoscopy showed that the left main bronchus was approximately normal to a point below the orifice of the left upper lobe bronchus, but below that point the lumen was narrowed to 1.5 mm., the walls were lined with granulation tissue which bled freely on instrumentation and the foreign body could not be directly visualized. Further effort to secure the nail by bronchoscopic approach was abandoned, and shortly thereafter the nail was recovered by a two-stage surgical approach. It was found lying in a cavity filled with foul pus. The postoperative course was protracted, moderately stormy and characterized by continuation of cough, purulent sputum and occasional hemoptyses.

In April, 1929, she still had a discharging sinus at the site of operation, and still complained of much cough and purulent sputum. Bronchography with iodized oil demonstrated bronchiectasis of the left lower lobe. In July, 1929, a multiple stage cautery lobectomy was performed. As this procedure progressed the involved lobe was found to be atelectatic, densely fibrosed, and filled with many small discrete abscess cavities. During this program one very severe hemorrhage occurred from the lung tissue. Fortunately it happened during a dressing and was promptly controlled. By September, 1929, she was able to leave the hospital, but not until January, 1930, was the wound entirely healed.

This patient is still living, married, and, when last examined in July, 1937, was free of any evidence of pulmonary disease. She reports continued good health in March, 1939.

No lung tissue was ever submitted for pathological examination in this case, but the surgical findings and the x-ray evidence leave no doubt as to the bronchiectatic involvement of the entire lobe. Her subsequent good health indicates that pathology was limited to one lobe.

CASE 2.—G. N., 23 months old, white boy, was seen early in March, 1931, because of cough, fever and a recently diagnosed metallic foreign body in the right lower airways. The baby had enjoyed good health until ten weeks prior to admission, when he had had a sudden unexplained "choking spell." His grandmother re-

lieved him by slapping him on the back and using "home remedies." Within a few days the baby became seriously sick and the family physician diagnosed his condition as "grippe." This "grippe" did not clear up but persisted for more than a month. The diagnosis was changed to "acutely inflamed enlarged tonsils," and immediate tonsillectomy was advised but not performed. A consultant saw the child and made a diagnosis of pneumonia with maximum involvement in the right upper lobe. Throughout his illness the child had been running an irregular temperature and had been coughing continually. The cough had been apparently productive, but, child-like, he had evidently swallowed all sputum. A roentgenogram was finally secured nine weeks after the "choking spell." It was a poor picture but did show a metallic foreign body in the right bronchus.

There was slight limitation of motion on the right side of the chest. Vocal fremitus was well transmitted over right upper lobe but was absent over right lower and middle lobes. Percussion note was hyperresonant over right upper lobe, flat over right lower and middle lobes. Breath sounds were harsh over right upper lobe, suppressed and accompanied by moist rales over right lower and middle lobes. All signs on the left were normal. Fluoroscopy showed a wire nail lying in the right stem bronchus, just below the bifurcation of the trachea, point up, head down, right upper lobe clear; density at right base; a suggestion of fluid in the right pleural cavity; left side of chest clear; heart in midline; mediastinum clear.

On March 10, 1931, the child was bronchoscoped, a 4 mm. tube being used. The point of the nail was found embedded in the wall of the right bronchus, just below the tracheal bifurcation. The field was obscured by continual outflow of pus. The nail was not recovered.

Bronchoscopy was followed by recurring attacks of dyspnea and cyanosis. After two such attacks, tracheotomy was performed. The respiratory difficulty was not relieved and the baby died early the next morning.

A limited autopsy showed:

1. Mediastinal emphysema.

2. A wire nail deeply embedded in the wall of the right bronchus.

3. Acute suppurative pneumonitis of right lower lobe, with multiple abscess cavities.

4. Acute suppurative pneumonitis of right middle lobe.

5. Dense pleuro-pulmonary adhesions throughout the right. (No fluid.)

CASE 3.—E. B., 26 years old, white, married housewife, was seen in October, 1931, because of a known open safety pin in the right main bronchus which she had aspirated three years previously. At the time she was suffering from "bronchial pneumonia," but had gotten out of bed to change her baby's diapers. She placed the small safety pins in her mouth, had a sudden cough paroxysm and aspirated one pin. She knew it, but not feeling any serious ill effects from it, intentionally let the matter drop. About one year after this accident she could feel a pricking sensation when she coughed and when she raised her arm. Otherwise there had been no pain or discomfort. From time to time she would cough up a plug of dried mucopurulent material which showed the impress of the keeper. There had never been any acute inflammatory reaction, pneumonia or pneumonitis. She had apparently remained in good health.

Physical examination failed to disclose any abnormal signs in the chest. Fluoroscopy showed no evidence of pulmonary inflammation. An open safety pin was clearly visible, apparently in the right main bronchus.

After a futile effort to recover the pin, the patient was referred to Dr. Jackson in Philadelphia, who easily removed the foreign body. Her convalescence was interrupted by acute pneumonitis of the right lower lobe and complicating empyema, which was drained at the Temple University Hospital.

Eventually she made an excellent recovery and safely passed through an appendectomy under ethylene anesthesia in August, 1934. There was opportunity to examine her chest from time to time through July, 1935, and at no time could any evidence of chronic pulmonary disease be discovered.

CASE 4.—(This case has been elsewhere reported by one of us and is here presented very briefly.)* H. B., 13 years old, white school boy, was seen in November, 1935, because of severe pulmonary hemorrhages. No history of foreign body could be obtained. A roentgenogram revealed slight infiltration in the right lower lobe.

*Butler, E. F.: Recognition of Radioparent Foreign Bodies at the Periphery of the Lung. Ann. Jour. Surg., New Series XXIX, 285, 1935.

Bronchoscopic examination failed to reveal a foreign body, but did prove the right lower lobe to be the source of hemorrhage. A therapeutic pneumothorax controlled the bleeding for four months. The hemorrhages then recurred with increased severity. A one-stage lobectomy was performed in March, 1934. In spite of multiple transfusions the boy failed to survive.

On examining the excised lobe, a foregin body, 1.75 cm. in length and about 2.0 mm. in diameter, was discovered in an abscess cavity near the periphery. The foreign body was macerated and soft. Microscopically it was undoubtedly of vegetal origin. Probably it was a little sliver of wood, possibly a grass stalk. The hemorrhage had come from the cavity within which the foreign body lay. In the immediate neighborhood there was noted hemorrhagic infiltration, fibrosis and bronchitis. The rest of the lobe appeared normal on both gross and microscopic examination.

We are indebted to Dr. Max Pinner,* Pathologist to the New York State District Tuberculosis Hospitals, for the following brief report of a case coming to autopsy, elsewhere, in 1927. This was, of course, before the widespread use of either bronchoscopy or lobectomy.

CASE 5.—A hospital orderly with a past history of empyema which, at one time, had been injected with a bismuth paste, was known to have chronic pulmonary suppuration. Roentgenograms invariably showed a dense shadow near the left hilum. This was interpreted as a remnant of bismuth paste. The patient was never bronchoscoped. On a program of postural drainage he was able to continue at work for some years in a hospital devoted primarily to the care of pulmonary tuberclosis. He finally developed a metastatic brain abscess and died. At autopsy a metal screw cap was discovered in the left lower lobe bronchus. This was the true cause of the shadow which had been interpreted as bismuth residue. Chronic suppurative bronchiectasis was found in the left lower lobe and was limited to that lobe. The brain abscess was, of course, secondary to the chronic pulmonary suppuration.

CASE 6.—E. C. D., 2½ years old, white, male child, was admitted to the Mount Morris Tuberculosis Hospital on September 23, 1936, because of cough, foul, purulent sputum, fever and loss of weight. Except for whooping cough a year earlier and a severe case of chickenpox half a year earlier, the past history was negative.

*Pinner, Max. Personal communication.

About August 1, 1936, the child, while following his mother in the barnyard, developed a severe choking spell. Just previously he had had a piece of timothy grass in his mouth. After five minutes of violent coughing he appeared to be all right, but the following night he became delirious and feverish, and from that time on remained intermittently sick. The possibility of foreign body was evidently overlooked, and he was treated for a variety of conditions. Cough continued and his breath became foul. A week prior to admission to the Mount Morris Hospital the patient coughed up a large amount of mal-odorous yellow material. By this time he was desperately sick. A second physician then obtained the history of the barnyard choking episode and promptly secured his hospitalization.

At the time of admission the child was acutely ill with a high temperature. Physical and roentgenological examination indicated an abscess riddled right lower lobe, pneumonitis involving the right middle and upper lobes and a right pyopneumothorax. The history predicated a retained foreign body. Bronchoscopy was deferred. First attention was directed toward the empyema, and on the date of admission simple intercostal drainage was instituted.

On October 6, secondary thoracotomy with rib resection was performed. A search was made for a free-lying foreign body in the pleural cavity, but none was found.

On October 27, bronchoscopic examination with a 5 mm. tube failed to reveal a foreign body but did disclose much thick, blood-stained mucopurulent secretion coming from the right lower lobe.

Within a month the pneumonitis of the right upper and middle lobes had cleared, the empyema cavity had almost obliterated but chronic suppuration persisted in the right lower lobe. The child was discharged November 11, 1936, with the recommendation that time be allowed for the recent acute processes to completely subside and the dense adhesions to thin out, in order that lobectomy might be more safely performed at a later date.

The patient was followed in the out-patient department and remained asymptomatic, except for occasional coughing and foul odor to his breath. Roentgenograms differed little from the one on discharge from Mount Morris until June 16, 1937, when increased density was noted in the shadows at the right base. Shortly thereafter the patient developed marked increase in cough and the temperature became elevated continuously to the vicinity of 101 de-

grees. On July 15 the patient developed persistent pain in the lower right thorax.

The child's second admission was to the Biggs Memorial Hospital on July 21, 1937. One-stage lobectomy (right lower lobe) was performed on July 27. On examining the lobe a timothy head was discovered in a bronchiectatic cavity. The postoperative course was stormy and characterized by bronchopneumonia in the opposite lung, foul discharge from the operative wound, and secondary anemia which required transfusion. Gradually the patient improved and he was discharged on September 17 with a small residual sinus at the site of the lobectomy wound which persisted until March, 1938. The child was last examined in February, 1939, and found to be well and free of any signs of residual pulmonary disease.

Dr. Max pinner has summarized the pathological findings in the excised lobe as follows:

1. Foreign body in bronchus.

2. Diffuse chronic pneumonitis involving the entire lobe with multiple small bronchiectases which are found in several bronchi, not only in the one that contains the foreign body. The pneumonitis consists mainly of diffuse purulent and cellular infiltration with relatively little organization. (The etiological agent for these changes has been found as the foreign body described above, which was identified by the Botany Department of Cornell University as belonging to the family of Timothy grass.)

CASE 7.—S. W., 11-year-old white school boy, was seen at the Homer Folks Tuberculosis Hospital, November 23, 1937, because of cough, bloody sputum and fever. On July 15, 1937, he had accidently inhaled a timothy head. This was followed immediately by a hacking cough and elevation of temperature to 102 degrees. The gravity of the situation was not immediately appreciated. One month later he began expectorating bloody mucus. In September a "pneumonic patch" in the right lower lobe was recognized. It had not cleared by November 23.

Bronchoscopy on December 10, 1937, revealed intense inflammation of the right lower airways and purulent secretions coming from the right lower lobe. No foreign body was seen. Fusiforms and spirochetes were demonstrated in the aspirated material. Surgery was advised but postponed until after Christmas.

On December 23 there was an hemoptysis of one-half cup of blood in which were found some timothy "beards."

He was readmitted to the hospital on January 14, 1938. His breath was now very foul. Physical examination, roentgenograms and bronchoscopy all showed an increase in the intensity of the suppurative disease at the right base. The patient was daily raising an average of 90 grams of purulent sputum, which contained very many spirochetes and fusiform bacilli. A period of bed rest with postural drainage and arsenical therapy was instituted as a presurgical measure. The patient showed marked improvement on this regimen.

On February 1, 1938, under avertin and nitrous oxide-oxygen anesthesia, a one-stage lobectomy (right lower lobe) was performed. The operation was difficult because of dense adhesions between the lung and the lateral chest wall and diaphragm. A transfusion of 500 cc. was given on the table. On examining the excised lobe a timothy head was discovered.

The postoperative course was complicated by severe infection of the lobectomy space. On February 8, *B. Welchii* was identified by the usual bacteriological tests. This complication was treated by thoracotomy, continuous oxygen flow through the thoractomy tubes, three doses of anti-toxin and multiple small transfusions. A severe but transitory serum reaction occurred on February 16.

On March 15 the patient's general condition having greatly improved, the whole lobectomy area was laid wide open for intensive antiseptic treatment.

Improvement was very rapid, and on March 30, 1938, this wound was closed, save for its lower edge. A bronchial fistula had developed, but this finally closed, spontaneously, during April, 1938. When last examined in February, 1939, the boy was well and free of any evidence of residual pulmonary disease.

Dr. Max Pinner has summarized the pathological findings as follows:

1. Foreign body in bronchus.

2. Chronic suppurative pneumonitis involving the entire lobe with multiple abscesses and marked organization.

CASE 8.—R. M. F., 1½ years old, white, male child, was admitted to the Biggs Memorial Hospital on June 11, 1938, because of fever, cough and gastrointestinal upset. On April 14, 1938, he had had a severe choking spell while chewing on spongy gum rubber eraser. The child's mother succeeded in removing several fragments

from his mouth. Vomiting was induced and more rubber was recovered. Within "a few days" he developed fever, vomiting and abdominal distention. Medical opinions were divided as to the cause of the symptoms. About April 25, 1938, there was sudden onset of cough. It was undoubtedly productive, but the child never expectorated the sputum. He vomited however, much purulent material. His breath became foul. A month after the initial choking spell, three "large pieces of the eraser" were passed by rectum, with some relief of the abdominal symptoms, but the fever and cough continued. Not until a week prior to admission were any roentgenograms of the chest secured. Pulmonary pathology was demonstrated and the child was referred to the hospital for further treatment.

On admission the child was acutely sick, somewhat dyspneic, emaciated, markedly anemic. The chest was asymmetrical, the left side being larger than the right. On the left, respiratory motion was limited, the percussion note was everywhere flat, vocal fremitus could be obtained only at the apex anteriorly, breath sounds could be heard over the upper half but were distant or inaudible over the lower chest. The right lung was clear. The heart was displaced to the right, the rate was rapid but regular. The temperature was 103.6 degrees, the pulse rate was 160, the respirations were 60 per minute. The red blood cells numbered 3,830,000; hemoglobin was 9.2 grams; white blood cells 15,950; differential, neutrophiles 47.5 (0/3/12.5/32), lymphocytes 40, monocytes 11.5, eosinophiles 1.0.

Roentgenograms, on admission, showed a diffuse shadow, with small areas of diminished density, occupying the entire left chest, deviation of heart shadow to the right, slight infiltration on the right. The diagnosis was acute pneumonitis with multiple small abscesses of left upper and lower lobes. Empyema on the left was also suspected.

On June 13, 1938, the child received a transfusion of 100 cc. of blood. Thoracocentesis on the left ruled out the possibility of empyema.

On June 16, 1938, bronchoscopy, with a 4 mm. bronchoscope showed pus and granular greyish-white foreign material within the lumen of the left main bronchus. The mucosa was swollen and forceps space was lacking. The foreign material was friable and crumbled under forceps pressure. Some was removed but certainly not all was recovered. There was a great outpouring of pus. The fragments of foreign material were examined and identified as rubber.

Roentgenograms, the next day, showed some clearing in the left upper lobe. The left lower lobe remained obscured. Another transfusion was given. The child remained desperately sick.

On June 19, 1938, tracheotomy was performed for impending asphyxia. The trachea was full of pus. The child died soon after. Necropsy was requested but not obtained.

The diagnosis, in this case, was based on the clinical picture, physical signs, bronchoscopic findings and roentgenograms. Acute suppurative pneumonitis due to obstruction of the left main bronchus by a foreign body was obvious. The degree of damage to lung tissue could only be surmised.

CASE 9.—C. M., 14-year-old white girl, was admitted to the Mount Morris Tuberculosis Hospital on October 14, 1938, because of cough, foul sputum and hemoptyses. In July, 1926, at the age of two, she had had a severe choking attack, while playing with small dried beans. The coughing soon ceased, but from that time on she was intermittently sick. Her sickness was characterized by bouts of fever and cough. She was said to have "pneumonia." In November, 1927, she was admitted to the Jefferson Hospital in Philadelphia under the care of Dr. Chevalier Jackson, who has kindly supplied us with the details of her hospitalization at that time.

During her hospitalization in Philadelphia, a series of fifteen bronchoscopic treatments was carried out. On one of these treatments the following note was made: "December 28, 1927. A small quantity of pus was removed from the right lower lobe bronchus. A small particle of material, probably of vegetal origin, was removed from the subdivision of the right lower lobe bronchus with the aspirating tube." The child was discharged April 21, 1928, greatly improved. The final diagnosis was "chronic suppurative bronchitis. Bronchiectasis right lower lobe bronchus."

The cough and expectoration of sputum continued until September, 1936, when she became asymptomatic, and so remained until December, 1937. At that time she contracted an acute respiratory infection. Cough and sputum recurred, the sputum becoming foul in January, 1938. In February, 1938, she had several hemoptyses, during which she expectorated 12 ounces of bright red sputum. She was in bed about six months. Cough and expectoration of foul-smelling sputum continued. Again, in August, 1938, she had a hemoptysis of four ounces. She was referred by her family physician to the out-patient department, at which time a diagnosis

of chronic bronchiectasis and atelectasis of right middle lobe was made.

On admission to Mount Morris Tuberculosis Hospital, physical examination showed: "Right lung—dullness below the fourth anterior rib and seventh dorsal spine. Breath sounds diminished anteriorly and diminished to distant posteriorly. Vocal and tactile fremitus diminished throughout. A few coarse rales below the angle of the scapula. Left lung—no abnormal findings elicited. There is slight clubbing of the fingers. Tonsils are enlarged." Roentgenograms: "Right lung—light to marked density below the level of the fourth anterior rib, with the upper margin of this density being represented by a straight line. Left lung—no intrapulmonary pathology visualized." No significant laboratory findings were reported.

The tonsils were removed under local anesthesia on November 21, 1938. The maxillary sinuses were drained under light general anesthesia on January 18, 1939. The right middle and lower lobes were removed by two-stage lobectomy (March 16, 1939, and April 18, 1939). Convalescence has been very stormy but ultimate recovery seems assured.* The excised lobes were examined by Dr. John Loesch, pathologist to the hospital. No foreign body was discovered.

Pathological diagnosis was: Chronic bronchitis with bronchiectasis.

PATHOLOGY

TABLE II

Hemorrhage	5
Pneumonitis, suppurative, acute (Total lobes involved, 8)	4
Pneumonitis, nonsuppurative, acute	1
Bronchopneumonia, contralateral	2
Abscess, pulmonary, localized	1
Bronchiectasis, suppurative, chronic (Total lobes involved, 6)	5
Bronchostenosis	1
Empyema, acute and chronic (Does not include post-lobectomy empyemata)	3
Abscess, brain, metastatic	1
Infection with gas bacillus	1
Emphysema, mediastinal	1

*Patient discharged from hospital, cured, during August, 1939.

Table II enumerates the various pathological conditions, caused directly or indirectly by foreign bodies, which were encountered in these nine patients. Hemorrhage, although properly a symptom, has been included in the list. First, the several conditions will be considered; then groups exhibiting similar clinical pictures will be discussed.

Table III sets forth the details of the five cases (55.5% of the series) in which pulmonary hemorrhage occurred. In the four bronchiectatics, hemoptysis was never severe nor was it of frequent recurrence. On the other hand, the case with the localized parenchymal abscess suffered from severe and frequent bleeding and finally succumbed to the effects of eleven massive hemorrhages occurring within three days. If lobectomy had been a first resort instead of a last resort, the result might have been different.

In determining the cause of hemoptysis, foreign bodies must be considered. It must be realized, also, that pulmonary bleeding facilitates the spread of infection to other lobes.

Table IV sets forth the details of the four cases (44.4% of the series) in which acute suppurative pneumonitis was observed. It is tragic to note that these are the cases in which delay had occurred in spite of clear-cut histories. Suppurative pneumonitis was an early event in the evolution of pathology. The process was invariably limited to the side on which the foreign body had lodged, but only once was it limited to a single lobe. It was the cause of death in the two fatal cases. The severity of the process was not governed by the degree of bronchial obstruction; the final result was, however, affected; patients with marked obstruction died, those without it survived. The further evolution of the process was followed in the two surviving cases. Lobes whose bronchi remained to some degree obstructed became the site of chronic bronchiectasis; lobes whose bronchi were not obstructed recovered from the acute process and have since remained free of disease.

Two of these cases presented physical signs and roentgenological findings which led to the suspicion of empyema or pleural effusion on the involved side. Autopsy in one instance, thoracocentesis in the other, disproved the suspected complication. Shaw* has shown that check valve obstruction may cause a suppurating lung to dis-

*Shaw, Robert R.: Acute Bronchiectatic Abscesses Simulating Empyema. Journal of Pediatrics.

TABLE III
HEMORRHAGE

Case	Foreign Body	Hemorrhage During Residence*	Hemorrhage After Removal†	Severity of Hemorrhage	Pathology Present At Time of Hemorrhage	Final Result
1	Wire nail	15 years	.....	Moderate	Bronchiectasis, left lower lobe. Bronchostenosis, left lower lobe bronchus	Cautery lobectomy Well
4 1933 / 1934	Sliver of wood	First symptom / 5 months	.....	Moderately severe / Severe and frequent	Localized abscess containing foreign body	Lobectomy Died
6	Timothy head	3 months	.....	Slight	Pneumonitis, acute, subsiding. Bronchiectatic abscess containing foreign body	Lobectomy Well
7	Timothy head	5 months	.....	Moderate	Pneumonitis, acute. Bronchiectatic abscess containing forign body	Lobectomy Well
9	Bean	.....	12 years	Moderate	Bronchiectasis, chronic, right lower and middle lobes	Lobectomy Convalescing

*Time after entrance.
†Interval after removal.

TABLE IV
Acute Suppurative Pneumonitis

Case	Age	Foreign Body	Location of Foreign Body	Degree of Bronchial Obstruction	Duration of Residence	Lobes Involved	Other Pathology	Bronchoscopy immediately performed	Final Result
2	2 yrs.	Wire nail	Right main bronchus	Almost complete	10 weeks	Right lower and middle lobes	Mediastinal emphysema	Yes	Tracheotomy Died
6	2½ yrs.	Timothy head	Right lower lobe (peripheral)	Negligible	2 months	Right lower middle and upper lobes	Putrid pyopneumo-thorax, right	No	Empyema drainage 1936 Lobectomy 1937 Well
7	11 yrs.	Timothy head	Right lower lobe (peripheral)	Negligible	5 months	Right lower lobe	None at time	Had been done earlier	Lobectomy Well
8	1½ yrs.	Rubber	left main bronchus	Complete	2 months	Left upper and lower lobes	None at time	Yes	Tracheotomy Died

tend and displace the mediastinum to the opposite side. The findings in Case 8 of this series conform closely to those reported by Shaw.

Acute nonsuppurative pneumonitis occurred once. It followed the bronchoscopic removal of a nonobstructing foreign body, but bronchoscopy had been previously performed and the trauma of repeated instrumentation, rather than the foreign body itself, may have been the responsible factor. The bronchi were patent and the acute pulmonary process subsided rapidly, but not without a complicating empyema which required surgical drainage. Subsequent examinations of the patient over a period of four years showed no residual pathology.

Localized abscess occurred but once. This is in sharp contrast to the diffuse lobar or multilobar pathology observed in the majority of cases. This abscess formed around a vegetal foreign body, which had rapidly passed to a peripheral position and there remained for five and a half months. The limited pathology in this case was offset by the frequent and severe hemorrhages from the walls of the cavity. The exsanguinated patient died after lobectomy.

Contralateral bronchopneumonia occurred twice. Once it complicated an acute suppurative pneumonitis during the residence of the foreign body. The patient died. Again it occurred after lobectomy, but was not of serious moment. That patient survived. The significance of contralateral pulmonary pathology will depend upon the degree to which it cripples remaining respiratory function.

Table V sets forth the details of the five cases (55.5% of the series) in which chronic bronchiectasis was demonstrated by roentgenological or pathological examination. In four cases the bronchiectasis was limited to the lobe invaded by or obstructed by the foreign body. In the remaining case two lobes were involved, neither of which was obstructed at the time of examination, the foreign body having been eliminated. In three cases the evolution of the bronchiectasis was watched over periods of two and a half months to eleven years. Two of these patients were seen during the course of the acute suppurative pneumonitis which occurred soon after the entry of the foreign body. The shortest interval from time of aspiration to pathologically proven bronchiectasis was five and a half months. It is reasonable to believe that every case which developed bronchiectasis had passed through an acute phase comparable to those actually observed. The other case had recognized bronchiectasis of the right lower lobe in 1927, fifteen months after the

TABLE V
CHRONIC BRONCHIECTASIS

Case	Age	Foreign Body	Location of Foreign Body	Degree of Bronchial Obstruction	Duration of Residence	Lobes Involved	Other Pathology	Pulmonary Hemorrhage	Final Result
1	19 yrs.	Wire nail	Left lower lobe bronchus	Almost complete	16 years	Left lower lobe only	Bronchostenosis	Moderate	Cautery lobectomy Well
5	? yrs.	Metal screw cap	Left lower lobe bronchus	Complete	? years	Left lower lobe only	Ancient empyema Brain abscess	None	Died
6	3½ yrs.	Timothy head	Right lower lobe	Negligible	1 year	Right lower lobe only	Previous acute suppurative Pneumonitis Previous empyema Post-lobectomy empyema	Moderate	Lobectomy Well
7	11 yrs.	Timothy head	Right lower lobe	Negligible	5½ months	Right lower lobe only	Recent acute suppurative pneumonitis Gas-bacillus infection post-lobectomy	Moderate	Lobectomy Well
9	14 yrs.	Bean	Right bronchus, unknown level	None	Had been removed 12 years previously	Right lower and middle lobes	Post-lobectomy empyema	Moderate	Lobectomy Convalescing

foreign body had been aspirated and spontaneously eliminated; in 1938 there was bronchiectasis of the right lower and middle lobes. Mild symptoms which had persisted for eleven years, in spite of a series of competent bronchoscopic treatments, suddenly became aggravated and alarming. One bronchiectatic died of brain abscess. The other four were treated by lobectomy and all survived, although, in each instance the postoperative convalescence was stormy.

Bronchostenosis occurred but once. Such stenoses are frequently the result of a foreign body, which, in this case, was a wire nail, retained for presumably sixteen years in the left lower lobe bronchus. The main bronchus was not involved, but the lumen of the affected area was reduced to a diameter of 1.5 mm. Cautery lobectomy was performed in 1929; the patient is still living.

Empyema, a recognized complication of pulmonary suppuration, occurred in three instances. Post-lobectomy empyemata are not included in this summary. In one case an acute putrid pyopneumothorax was noted within two months of the foreign body accident. The air in the pleural cavity was not under tension. Simple drainage was promptly instituted and the patient survived. Another acute empyema occurred as a complication after an otherwise successful removal of a nonobstructing foreign body. The patient was drained and has remained well. In the third case the empyema was on the same side as the foreign body, but was in the chronic stage when observed. It is impossible to state whether it was caused by the foreign body or not.

Mediastinal emphysema was found once at autopsy. It might have been caused by tracheotomy, bronchoscopic trauma or by the foreign body, a wire nail whose point had punctured the right main bronchus.

Brain abscess occurred once. It was the terminal complication in bronchiectasis due to a retained but unsuspected foreign body.

Gas bacillus infection occurred once, as a most unusual post-lobectomy complication. The bacilli were undoubtedly carried into the lung by the foreign body, a timothy head. The patient survived.

DISCUSSION

These nine cases may be regrouped as follows: nonobstructing foreign bodies; obstructing foreign bodies associated with fulminating acute suppurative pneumonitis; obstructing foreign bodies associated with chronic suppurative bronchiectasis; peripherally re-

tained foreign bodies. Such an arrangement will better correlate location and obstructive effect of the foreign body, resulting pathology and treatment.

The one instance of nonobstructing foreign body will serve as a foil to the other cases. After three years of residence it had caused negligible pathology. This is in sharp contrast to the extensive pathology caused, within a few weeks, by obstructing foreign bodies regardless of their nature. Bronchoscopic removal of a nonobstructing body should suffice. The bronchus, being patent, would permit adequate drainage of all secretions, if inflammatory reaction occurred.

There were two cases with bronchial obstruction and coincident fulminating acute suppurative pneumonitis. Both were bronchoscoped with poor results as to removal of the foreign body. Both developed respiratory difficulty, both had tracheotomy with little relief to their dyspnea, both died. Two additional patients were seen during the course of acute suppurative pneumonitis, but without significant bronchial obstruction, the foreign body having passed to smaller subdivisions of the lobe bronchus. One of these cases had, in addition to the pneumonitis, a putrid empyema. Neither of these patients was bronchoscoped until the acuity of the condition had subsided. Both recovered.

What treatment should be planned for cases that are desperately sick at the time of admission? Should an immediate effort be made to remove the foreign body, in the belief that restoration of bronchial drainage is imperative? Should an expectant policy be adopted, with reliance on postural drainage, supportive measures and good nursing care to tide the patient over the acute phase? Radical surgery is obviously out of place. It is a most difficult situation, and each case should be decided on its individual merits. Conservatism would seem to be the wiser policy, unless the patient arrives in the hands of one who has had an extraordinarily large experience in foreign body problems.

Two cases of obstructing foreign body associated with chronic bronchiectasis have been observed, as well as three additional chronic bronchiectatics without significant obstruction. In two of the latter three the foreign body was still in situ, in one it had been eliminated twelve years previously. One obstructing foreign body caused death by metastatic brain abscess. Another obstructing foreign body was removed by surgical approach, drainage was established, but the bronchiectasis was not cured. In another instance a series of expert

bronchoscopic treatments, after the elimination of the foreign body, failed to prevent the later spread of bronchiectasis to an adjacent lobe. Four of the five bronchiectatics eventually came to lobectomy and all survived.

The problem presented by obstructing foreign bodies associated with chronic suppuration is simpler. These patients have survived the acute fulminating phase of their infection. They have re-established some degree of bronchial drainage. Their drainage would be vastly improved by removal of the foreign body, but even so, there would remain irreparably damaged lung tissue. It would be futile to wait long for the result of foreign body removal to become apparent, because the bronchiectasis could not be cured by time, by local medication, by radiation, by drainage, by any measure of so-called collapse therapy. Palliation could be secured, but not cure. Cure demands complete excision of all involved lobes. The soundest policy would seem to be: removal of the foreign body if it be easily accessible; an interval of general care, including bronchoscopic treatment and postural drainage, and then, granting that there is no obvious contraindication to radical surgery, lobectomy at the earliest reasonable opportunity.

Three patients with peripherally retained foreign bodies have been observed. In none could the foreign body be visualized, either by roentgenogram or by bronchoscope. Two patients had established bronchiectasis, the other a localized abscess cavity and severe hemorrhage. The optimum policy in similar cases would seem to be, disregard of foreign body and lobectomy at the earliest possible moment.

SUMMARY

Particular emphasis has been placed upon two pathological conditions, acute suppurative pneumonitis and chronic suppurative bronchiectasis. One or both of these conditions were observed in seven of the nine patients. It is reasonable to believe that they are evolutionary phases of the same entity. It is also reasonable to believe that chronic suppurative bronchiectasis, with or without bronchostenosis, is the probable fate of any patient who has experienced and survived prolonged obstruction of the airways by aspirated foreign body. If these premises be granted it becomes apparent that, once pulmonary suppurative has become established, removal of the foreign body is no longer adequate to prevent or cure chronic disease. The extirpation of the damaged lobe becomes a requisite for cure.

CONCLUSIONS

1. There is still need for education of the profession and the laity regarding the dangers due to aspirated foreign bodies.

2. Prompt diagnosis and prompt removal of foreign bodies are of the utmost importance.

3. Prolonged residence, without bronchial obstruction or trauma to lung parenchyma, may be well tolerated.

4. Prolonged residence with bronchial obstruction is not well tolerated and leads first to acute suppurative pneumonitis, later, if the patient survive, to chronic bronchiectasis.

5. Acute suppurative pneumonitis carries a high mortality. Bronchoscopic efforts are not well tolerated.

6. Bronchiectasis is incurable save by radical surgery. Therefore, not only should the foreign body be removed, but also the irreparably damaged lung tissue should be removed by lobectomy.

Biggs Memorial Hospital.

Society Proceedings

CHICAGO LARYNGOLOGICAL AND OTOLOGICAL SOCIETY

Meeting of Monday, March 6, 1939

THE PRESIDENT, DR. THOMAS W. LEWIS, IN THE CHAIR

Laryngoptosis

J. E. MULSOW, M.D.

MILWAUKEE

(This paper is printed in full on page 779 of this issue)

DISCUSSION

DR. SAMUEL SALINGER: When I saw the title of Dr. Mulsow's paper I had a feeling that I had read somewhere in some journal about a similar case. I looked it up and found it and now I want to tell you about it because the question of trauma was brought up by Dr. Mulsow. The case to which I refer was reported by Langenbeck in the Zeitschrift. A man was adjusting the bridle on one of a team of horses when the animal became unruly and lunged forward. The chain running from the bridle to the pole caught him in the neck, forcing him against a tree. He was knocked unconscious, vomited and expectorated blood. For some time afterward the patient had difficulty in swallowing, became emaciated and had a cough due to lack of control of the larynx in the act of swallowing. X-ray pictures revealed the thyroid cartilage separated from the hyoid and displaced downward for a distance of about three fingers breadth. Langenbeck states that he was able to find only one similar case in the literature. In Dr. Mulsow's case it seems to me that had the disclocation been due to trauma there should have been more symptoms such as edema, hemorrhage, etc., since the act of tearing the hyoid bone from the base of the tongue must necessarily involve much force. As for congenital dislocation, the cases are so rare that very little is known of the etiology.

Therefore, when a case of this type is encountered, particularly if there is a question of liability or compensation, it is important to know that at least two undoubted cases due to trauma have been reported and are a matter of record.

DR. PAUL HOLINGER: We have had two cases recently in which it may be said that laryngoptosis was due to trauma. In the first case, the patient had been lifted sixteen feet off the ground by a belt which wrapped itself around his neck. In the second case, the patient had been playing "Indian." In both cases the thyrohyoid ligament was torn, but in neither case was there a history of hemoptysis.

DR. J. E. MULSOW (closing): It seemed evident that this man had no difficulty in swallowing. The movements of the head and protrusion of the tongue were normal. There was no fibrosis, the tissues of the neck were smooth and soft. It appeared to be of congenital origin.

Spontaneous Hemorrhage From the Ear

T. F. MCCORMICK, M.D.

MILWAUKEE

(This paper was published in full on page 252 of the June issue)

DISCUSSION

DR. SHERMAN SHAPIRO: While I personally approve of Dr. McCormick's treatment, I still wonder how the inferior and superior petrosal sinuses, which would be below the site of the tamponade, would be controlled in this case.

DR. HOWARD BALLENGER: Did you find any evidence of a dehiscence in the bulb in this case?

DR. JOSEPH C. BECK: I wonder if Dr. McCormick has come across the report of Dr. M. A. Goldstein of St. Louis, in which he called his case vicarious spontaneous bleeding, because there really was spontaneous bleeding in the region of the bulb during menstruation. The blue color could first be seen through the tympanic membrane. At first there was bleeding into the pharynx and subsequent dissolving of the drum membrane and bleeding externally that required packing. It was possible to stop it for a time, but

once it did not stop and the patient bled so that venous ligation was necessary, including blocking off of the lateral sinus.

I do not think these cases are limited to children altogether. I think the cases you recall from Politzer are advanced cases of tuberculosis osteitis, and I think that is what many of them are.

DR. SAMUEL SALINGER: I think that Dr. McCormick's procedure in his case was logical. He was mindful of the fact that profuse hemorrhage from the ear may be due to erosion of the carotid artery. In his case, however, there was no evidence of any infection in the throat from which an involvement of the carotid artery might be expected. On the other hand, it is well known that erosion of a vein is extremely rare except as a result of fulminating virulent infections. Nevertheless, in the presence of an ear infection and absence of any throat swelling, he was forced to expose the sinus and pack it off. Since this procedure stopped the bleeding, it is safe to assume that the bleeding originated from the jugular bulb. However, it is impossible to make this assumption a positive fact without direct exposure and inspection of the bulb.

DR. T. F. MCCORMICK (closing): I think Dr. Salinger has summed it up pretty well. Concerning Dr. Shapiro's question, it is true that the sinus was collapsed at a point above the entrance of the inferior petrosal, but nevertheless, the bleeding did not recur. Dr. Beck is right about Politzer's cases; these were all in suppurative processes of the ear of long standing. The twenty cases in children were reviewed by Doctors Pearlman and Salinger. As for any dehiscence in the bone, I do not know; I performed the usual mastoidectomy, exposed the sinus widely, and packed it off.

Rhinitis and Sinusitis Caseosa

ORLANDO P. SCHOOFS, M.D.

MILWAUKEE

Rhinitis or sinusitis caseosa is an uncommonly observed condition characterized by an accumulation, within the nose or paranasal sinuses, of a grayish-yellow to brownish, glistening, occasionally fetid, putty-like exudate. Microscopically the caseous masses are structureless, containing degenerated polymorphonuclears and fatty acid crystals. Bacteriologically various organisms may be found, predominantly fusiform bacilli, spirochetes, small filamentous growths and various strains of staphylococci and streptococci.

According to Ballacs, three types occur clinically:

1. A form accompanied by mucosal hypertrophy and polypoid degeneration.

2. Another in which periostitis accompanies deep inflammatory processes of the accessory sinuses, occasionally with bony necrosis ending in spontaneous rupture.

3. Finally, one in which there is rhinolithiasis, atrophy and ulceration. Caseous rhinitis must be distinguished from apparently similar conditions occasionally seen in the presence of tumors, foreign bodies, tuberculosis or lues.

Widely diverse opinions are held as to its cause and mode of development:

1. Duplay, who first described it in 1874, observed two cases following facial erysipelas, and considered them due to extension, intranasally, from the external lesion.

2. Cozzolino's idea was that it is based on a scrofulous diathesis, but this was also quickly rejected.

3. Another opinion is that it is a disease entity. Various organisms were held responsible, especially the streptothrix alba. Masini stated that the disease is too decisive a clinical personality not to be motivated by a specific undetermined organism.

4. Numerous clinicians, especially Bruzzone, who reported 18 cases, believed it to be a nasal cholesteatoma; this theory was quickly discarded because of the absence of concentric lamellæ, a diseased matrix and other characteristic features.

5. Foreign body as an irritating factor.

6. Necrobiosis of a polyp.

7. That bony necrosis was responsible for the character of the secretion. This lacked supporting evidence, because sequestration rarely occurs, and roentgenologically sclerosis is found as well as osseous rarefaction.

8. The most commonly accepted explanation has been that an underlying nasal deformity in the presence of chronic suppurative sinusitis causes retention of discharges which undergo physical and chemical transformation.

Avellis concurred in the opinion that metamorphosis of an empyema takes place in consequence of the closure of an ostium;

the caseous mass later acts as a foreign body which finally may cause bony absorption.

Ballacs in 1936 (Monatschrift) formulated a similar conclusion and held that several factors were necessary:

1. A dystrophic condition of the mucosa, the result of chronic inflammation.

2. Stasis caused by nasal obstruction.

3. The transformation of secretion by unascertained micro-organisms.

Myersberg et al., in an article appearing in the Archives of Otolaryngology, April, 1936, also temporarily accepted the popular belief of a physiochemical change taking place in the stagnating secretion produced by chronic sinusitis.

Greifenstein in the last issue of the Zeitschrift reported the results of his investigation of spontaneously arising and experimentally produced caseous rhino-sinusitis in rabbits. Both macroscopically and microscopically there was an absolute conformity in all stages and forms of the spontaneous type in rabbits with the clinical picture observed in human beings. Microscopically the experimentally produced condition in rabbits could not be differentiated from the spontaneous type. Primary or secondary atresia of the ostia was seldom present, nor could the process be in any way considered a form of pyocele; the assumption of a caseous metamorphosis is entirely untenable.

Histologically the inflammatory reaction produced by chemical irritation is not a pseudomembrane but consists of an intense subepithelial leucocytic infiltration, which may only implicate a portion of the inner surface of the sinus wall. Necrotic loss of the epithelium may occur.

He concluded that caseous rhino-sinusitis must be considered an entity which cannot be regarded a form of rhinogenous parasinus infection.

Wirth, in discussing Greifenstein's treatise, said that he believed gram negative cocco-bacilli were instrumental, and he could by innoculation regularly produce the condition in rabbits and guinea pigs.

The prognosis is excellent; complete removal of the pultaceous debris, and routine after-care results in a healthy nasal cavity with complete amelioration of symptoms with no tendency to recurrence.

REPORT OF CASES

CASE 1.—N. S., No. 2668. On March 12, 1931, I saw a pale, undernourished 32-year-old man, who had complained of shortness of breath and complete bilateral nasal obstruction for more than 10 years. Polypi had repeatedly been removed from both nasal cavities, but never with even the slightest partial restoration of any pernasal passageway. On examination both cava nasi were completely filled with mucous polypi.

Five days later (on March 17, 1931) a large number of pedunculated polypi were removed from both nasal fossæ. A marked mucosal hypertrophy and extensive polypoid degeneration of the structures of the left nasal wall was noted; this area was completely asthetic.

On March 19, 1931, additional pedunculated polypi were removed from the posterior portion of the right nasal cavity. Piecemeal removal by punch forceps of the hypertrophic tissue from the left nasal wall revealed discrete, grayish-yellow, pultaceous masses in the interstices between the polypoid excrescences, beneath the middle turbinate, and in the anterior and posterior ethmoidal cells. There was an entire absence of any secretion of a purulent nature.

He did not return for three months (June, 1931) because of a greatly improved sense of well-being and ability to breathe through his nose. There was, however, an obstruction to expiration through the right side when closing the left nostril. A postnasal polyp was removed from this side; also on two subsequent occasions.

November 23, 1931: Puncture-cautery of hypertrophic posterior end of lower left turbinate.

April 19, 1933: A moderately sized, fairly firm mass of tissue was removed from the posterior portion of the left inferior turbinate, which on biopsy was found to be constituted of loose connective tissue exhibiting chronic inflammatory reaction.

He has been perfectly well and is able to breathe through both sides of his nose. He has gained thirty pounds in weight. The left side, that which was implicated by the caseous process, is entirely clear. Two small polypi are present in the right side of his nose, extruding from the middle meatus.

CASE 2.—M. S., No. 2066. A 34-year-old woman was seen at her home on May 31, 1929. For three months she had had a constant severe headache associated with vertigo and occasional vomiting. For one week (during the last week of the first month of her illness) she was unconscious and delirious. All teeth had been extracted. The paranasal sinuses had been rayed and reported negative for evidence of infection. A diagnosis of gastric ulceration had been made and operation advised. Asthenia was marked and emaciation apparent (weight 65pounds, usual 95 pounds). (An instructor in a high school to whom the x-ray plates, which were taken in March, were shown said he thought the sinuses were infected.)

On examination the septum was found deviated to the right with marked engorgement of the left turbinate bodies. A small amount of thin, foul-smelling seropus was present on the nasal floor. The ears, throat and the function of the extraoculur muscles were normal. The x-ray plates showed a general blurring of the ethmoidal region and the left antrum, but were satisfactory in regard to the sphenoid sinuses.

One month later, in June, she was brought to my office. She could not walk without support. The headache, chiefly referred to the vertex and occiput, was still constant. After cocainization and shrinkage, pus was seen on the posterior portion of the left inferior turbinate. The left antrum was irrigated; the washout contained only a small amount of flocculent secretion, but no pus. A formed mass of pasty secretion was visible in the spheno-ethmoidal region. An attempt to remove this by a simple canular irrigation was unsuccessful. Several days later this mass was successfully removed by irrigation after repeated fragmentation by means of a dull ring curette. The fetor was practically unbearable. A relatively large opening was discovered in the anterior sphenoidal wall, the result of bony erosion. The cavity of this sinus was filled with a sticky exudate which was removed in a similar manner.

Recovery occurred promptly, with complete relief from her distressing symptoms and rapid improvement of her general health. She gradually regained the weight which had been lost. There has been no recurrence.

DISCUSSION

DR. ALFRED LEWY: We have all seen cases associated with sinus trouble in which the discharge was cheese-like in consistency, and I do not think I would know how to distinguish a case of rhinitis caseosa. I would like to ask what the distinguishing characteristics are, and what was shown by microscope. I am sure we have all seen the discharge vary all the way from watery to thick and cheesy in consistency, without diagnosing rhinitis caseosa.

DR. M. REESE GUTTMAN: Dr. Schoofs has, unfortunately, stolen my thunder. I was to present this subject before the Society last November, but due to my absence in Europe, the date of my presentation was changed to the forthcoming April meeting. So I believe, Mr. Secretary, you will have to withdraw my name from the program.

We have had experience with three cases. The first was seen by Dr. Beck in 1922 with a bilateral polyposis. He was operated upon and was not seen until twelve years later, when he was sent by his physician with a diagnosis of carcinoma of the antrum. He had a large brawny swelling of the left cheek associated with fixation as well as protrusion and deviation of the eye. The wall of the left nasal chamber was distended to such an extent that it pushed the septum into contact with the opposite or right nasal wall. X-ray disclosed a mass within the antrum. A tentative diagnosis of carcinoma was made and he entered the hospital for sublabial operation with the intention of resecting the growth and introducing radium. To our surprise we found, instead of a tumor mass, a large amount of foul, thick, cheesy debris. The antrum was cleansed and a large opening was made into the inferior meatus.

The patient made an uneventful recovery. Microscopic examination of the material showed no organic structure whatever. Bacteriologic report showed a large number of streptococci and staphylococci.

The second case was one in which there was a history of repeated recurrences of polyps in the nose after surgery. A radical sublabial operation also disclosed the caseous condition.

The third case was interesting because it showed the short period of time in which a caseous sinusitis might develop. Just before I left we had occasion to see a woman with bilateral polyps associated with nasal discharge and chronic bronchitis. X-ray findings indicated extensive involvement of the antra as well as the ethmoids. She was subjected to an extensive resection of the polypoid membrane in the ethmoids and antrum, the latter by sublabial incision. The findings at operation consisted of a thick hyperplastic polypoid mucosa associated with polyps. No evidence of any caseous material was noted at this time. She apparently underwent an uneventful postoperative course. Upon my return four months later I found that she had been seen three months after operation with a swelling of the left side with a fistula into the skin of the cheek. X-ray showed that the left antrum was blocked out. Reoperation sublabially disclosed the entire antrum filled with this caseous structureless foul material.

Histologic examination of each of these three cases disclosed no organic structure whatsoever, and bacteriologic examination disclosed numerous streptococci and staphylococci.

DR. JOSEPH C. BECK: What has become of the old term rhinitis pseudomembranaceous with caseous formation? That was a very common clinical entity in some of the European clinics, and it was not mentioned this evening.

DR. WALTER H. THEOBALD: I would like to inquire if any bacterial growth was found on culture from the discharge?

DR. O. P. SCHOOFS (closing): The diagnosis is based on the discovery of a caseous secretion in the absence of a tumor, tuberculosis or lues. The secretion found is entirely homogeneous, occasionally containing degenerated polymorphonuclears.

Greifenstein was able to produce the condition in rabbits by injection of various chemicals, principally turpentine. The lesion produced was not a pseudomembrane but consisted of an intense subepithelial leucocytic infiltration which did not necessarily involve the entire lining wall of the sinus. Greifenstein formulated his

conclusions and said that further investigation must be made by the clinician. Wirth, in discussing Greifenstein's report, said that he was able to produce a caseous rhinitis in rabbits and guinea pigs by inoculation with cocco-bacilli of the pseudo-tuberculosis type.

No cultures were taken in my cases. I believe no definite conclusions can be drawn from the literature, although Greifenstein's work is rather convincing.

Injuries to the Sigmoid Sinus

E. BACH, M.D.

MILWAUKEE

(This paper is printed in full on page 790 of this issue)

DISCUSSION

DR. ALFRED LEWY: In going over an experience of more years than I like to admit, I remember in my own experience of opening the lateral sinus, or having it open twice, unintentionally. In both cases there was a parasinus abscess. In the first case the sinus opened when I was wiping off the granulations with gauze around my forefinger. In the second case the sinus opened while I was washing blood off my glove, and I do not know how it happened. The wall was evidently necrotic. I have seen it accidently opened at operation when I was present—not operating—and I would say that the safest instrument is a sharp gouge and a mallet. I have seen sinuses opened secondarily after using a burr, and have seen them opened accidently by a curette and by a rongeur. During the early stage of operation when the operator had got to the sinus without having a wide-open space prepared first, it would constitute a difficult problem. I think it is important to work in a widely exposed mastoid field so that you can see what you are doing. I think the curette must be used very skillfully and I think the function of the rongeur is to remove the overhang and not the cellular tissue. If these rules are observed I think there will rarely be accidental opening of the sinus.

DR. SHERMAN SHAPIRO: I want to commend Dr. Bach on his courage in reporting a mishap which hardly anybody fails to encounter at one time or another. I think the way one looks upon this sort of accident depends upon our good fortune as to the consequences. I know of one case where an over-enthusiastic resident tore the sinus during operation, and the patient went on to a thrombosis and died in consequence of the accident. I would endorse

everything Dr. Lewy said, although I do not believe that any instrument is fool-proof, nor that under certain circumstances this mishap may not occur to anyone, due to a little lapse on the part of the operator or due to unforeseen circumstances. One thing at Research we have taught to residents, postgraduates and internes, which may minimize the danger, and that is to open the antrum and outline the trough between the posterior wall and the jugular first, before proceeding to the posterior part of the mastoid. In this way, if the sinus is accidently opened, you at least know that the major part of the operation has been accomplished and drainage established from the middle ear to the outside.

DR. ALFRED LEWY: I forgot to give all the details, and I would like to say that the method of procedure is the one advocated by Dr. Shapiro. I said to uncover the sinus first. I should have said first with the mallet and gouge, and you must be careful. The edge of the gouge is smooth, with no nicks. A nick is capable of causing serious damage. After having uncovered the sinus, after exposing the plate, the rest of the operation is done with the Lombard or Kerrison rongeur.

DR. JOSEPH C. BECK: I am suspicious that Dr. Bach presented this paper with the thought in mind that we all should confess our sins, and I will confess. I have opened the lateral sinus twice accidently, not in Chicago but in Cincinnati both times. Once in the City Hospital during the Ohio State Medical Society Meeting and once in the late Dr. John Murphy's practice. One was in an acute mastoiditis and the other a chronic suppurative ear in which I was doing a radical, I entered the lateral sinus. In neither of these cases did anything serious happen. I am sure many men have seen a slight wound in the lateral sinus, especially where there is not a streptococcic infection present and the patient is not very ill. By simply protecting the wound with vaseline gauze nothing will happen. In both of these cases I used the gouge, contrary to my usual procedure, which is the burr. I have always used the burr in my mastoid surgery. I can see the sinus much easier and more clearly, and if I want to expose the sinus then I follow the suggestion Dr. Lewy made. I call the Lombard Punch Beck's delight because it is the safest instrument I know to get to the sinus. This paper is a very learned paper, and everyone should get marked benefit from this method Dr. Bach mentions for entering the sinus. One should not get panicky because it has been accidently opened, and it does not, as a rule, require immediate packing off above and below, because many small rents do not go on to sinus thrombosis, but heal without further complications.

DR. L. BRACHMAN: This recalls a case that has nothing to do with operation, but was due to accident. My brother-in-law, about 30, received a fatal accident while dancing, when he fell and hit the left side of the head and had a very extensive fracture through the mastoid down to the base of the brain. He became cyanotic and within four minutes was dead. On postmortem, there was found a fracture and extensive bleeding toward the base of the brain, which caused death. There was a rent into the sigmoid which caused the bleeding.

DR. FRANCIS LEDERER: I believe Dr. Bach is to be congratulated for presenting the error of omission which he recounted. One might say that it has been generally thought that one could expose the sinus with more or less abandon. That is the conception Professor Neumann had, and I believe others. I believe it is rather a dangerous policy to assume that it does not make much difference whether you expose the sinus or not. Dr. Bach did not give any further details of technic, as to whether, when he punctured the sinus, he made a larger exposure. I believe it is rather good policy to expose the sinus further, perhaps having in mind that you can prevent complications and what he called attention to—there might be other bony spicules that become lodged between the bone and dura of the sinus.

The point of dressings is interesting to me, especially where you expose a perisinus abscess with granulations on the dura, which constitutes a weakened wall of the sinus. In such cases, especially in contagious practice, spontaneous or subsequent opening is not uncommon, whether the gauze catches on the granulations and weakens it still further, it is difficult to say. It might be well to use vaselinated gauze in packing. Recently there appeared an article in the Monatschrift, in which the author calls attention to a number of cases in which he exposed the lateral sinus and nothing happened, but I think it is bad policy to confidently assume that this will be universal. Dr. Beck rose in defense of the burr, but I assume that it is still a dangerous instrument around the lateral sinus, because there always remains a thin plate of bone beneath the burr, and it is that last thin layer of bone that requires the most cautious treatment.

DR. J. C. SCHOOLMAN: I wish to say a word about the reliability of the x-ray in regard to the position of the lateral sinus. We had an interesting case about two weeks ago. Examination of the film indicated an ample amount of room within the mastoid cavity, yet when the first piece of the cortex was removed there

was revealed what was apparently the sinus, and yet on re-examination of the x-ray we thought it must be pyogenic membrane. We decided to treat it as a sinus, and much to our surprise it was sinus so forward and lateral that the usual operation had to be somewhat modified.

DR. E. BACH (closing): I want to thank Dr. Lewy and Dr. Shapiro for their discussions, and I think they emphasized an important thing. I have seen mastoids with an extensive coalescence and breaking down, and if one proceeds with a curette he may have a deep cavity with steep overhanging walls. A sinus accident in a case like this would be a serious handicap. It would be hard to stop the hemorrhage and at the same time remove bone from the sinus above and below the injury. Dr. Lewy removes the mastoid cortex over a considerable area and then reaches the mastoid antrum, by means of a funnel-shaped excavation. This is safer, gives better visibility, and obviates difficulties in case of accident.

In regard to Dr. Lederer's question about what was done when the sinus was punctured, I wish to say that the bone chip measured about one-fourth inch in diameter. There was a small amount of bleeding. The chip was easily located and removed, but I did not make an extensive exposure of the sinus around the puncture. I was sure the chip was out, and when we put a small pack on the sinus it stopped bleeding. Of course, it is known that when the dura or lateral sinus is exposed, and a bone chip is pushed between the bony wall and left there, postoperative complications are likely.

Dr. Schoolman's case is very interesting, and is something we find occasionally. It is well to extend the incision forward when we encounter an anteriorly placed sinus, to expose the bony meatus and afford an anterior approach to the mastoid antrum.

Carcinoma of the Sphenoid and Ethmoids

T. L. TOLAN, M.D.

MILWAUKEE

DISCUSSION

DR. HOWARD BALLENGER: The first case reported by Dr. Tolan probably was primary in the nose or the nasopharynx. However, it would seem to me that the second case was metastatic in the nose secondary to some other focus.

A few years ago I had occasion to look up the reported cases of primary carcinoma of the nasopharynx, and if I remember correctly, regional metastasis, that is, metastasis to the cervical lymph nodes, occurred in most if not all the cases. However, metastases to the viscera seldom or never occurred from carcinoma of the nasopharynx.

Dr. Francis Lederer: It is always interesting to hear the history of a case of sinus malignancy, for one is seldom guided by the history, because the next case will run a different course. I would not feel badly about the subsequent course of the case if I were Dr. Tolan, because it would be difficult to anticipate in his case that a rectal carcinoma would metastasize there, although one might readily see that it could from the liver and the lung. This type of case is always of interest, because to my mind the nasopharynx is one of the very neglected fields of investigation in the course of routine examination. We have that exemplified on numerous occasions. Posterior rhinoscopic examination augmented by the use of the Yankauer is particularly valuable in visualizing this space for early diagnosis. I have in mind one instance of a patient who last July complained of pain in the neck and left ear. A cloudy antrum was found and thought to be the basis of his trouble. He complained of the neck also, but little attention was paid to that. There was a ptosis of the eyelid on the right side. Later on it became bilateral. Various diagnoses were not proved. He had been in the clinic for some time and not much attention was paid to the nasopharynx. One day I felt that inasmuch as Dr. Furstenberg, in reporting on a number of cases, had discovered that the nasopharynx was a neglected area, I carefully examined the nose and nasopharynx. I found a mass present which proved to be carcinoma. The specimen at autopsy was very interesting, inasmuch as we could trace the origin of the tumor invading practically the same area Dr. Tolan described in his second case. This case will be reported in detail at a future time. I have appreciated hearing this report on carcinoma, but I cannot feel that a biopsy stirred up metastasis. It would not have made much difference, as we see it now, but I think it is a possibility too dangerous to even admit, because we have sufficient difficulty in convincing lay people, let alone physicians, that it should be done. So I certainly would disregard that possibility and proceed in the next instance and try to obtain a biopsy.

Dr. Joseph C. Beck: I want to compliment Dr. Lederer on his discussion, particularly the attitude he takes toward biopsy. It

has always been our thought in teaching this regarding diagnosis, and I am glad he brought it out.

DR. T. L. TOLAN (closing): It was interesting in discussing with the group of general surgeons the case of metastasis from carcinoma of the rectum that no similar case had been seen or heard of. I have no thought that the biopsy stirred up metastasis in the liver and lung, as the symptoms and signs were evident too soon after surgery had been done.

Transverse Fracture of the Petrous Pyramid

W. E. GROVE, M.D.

MILWAUKEE

(This paper was printed in full on page 491 of the June issue)

DISCUSSION

DR. ALFRED LEWY: I would like to ask if there was any danger of facial nerve paralysis?

I would also like to say that these cases are usually the result of automobile accidents and later on lead to very difficult legal complications.

DR. PAUL CAMPBELL: The only thing I would like to bring out is the difficulty of trying to diagnose these fractures by x-ray. I have made a study of all fractures of this type I can get hold of, and the astonishing thing is that many do not show in the x-ray even though every care is taken. I recently saw a fracture of the petrous pyramid with gross evidence of a fracture line going to the outside, yet it did not show in the x-ray. The case was followed by autopsy after meningitis had developed several months later. There was a fracture in that very place, although it had not shown in the x-ray. I would like to ask what percentage you feel can be shown by x-ray?

DR. W. E. GROVE (closing): Dr. Lewy spoke of facial paralysis with this particular type of injury. It happens that none of these cases had facial paralysis. However, from a study of a mass of material and study of the literature, I can say that approximately 50 per cent of labyrinthine fractures are complicated by facial paralysis, and when a facial paralysis so complicates the fracture, it is apt to remain permanent, more so than the facial paralysis that occurs

after longitudinal fractures, although even the paralyses after transverse fractures show a large percentage of recoveries.

Dr. Campbell spoke of the difficulty in getting satisfactory x-ray pictures of these cases. I would say that the difficulties in showing basal fractures by x-ray are much greater in the longitudinal type, which is a middle fossa fracture running along the anterior border of the pyramid, than in the transverse type of fracture. If the x-ray men will take pains to view these fractures in a number of different positions, they can be shown. Stenvers' position is the best to use for the determination of labyrinthine fractures, because it exposes the pyramid excellently. Sometimes taking a picture at a little different angle will bring out the fracture; sometimes when we suspect it is there it cannot be shown by x-ray. However, inasmuch as there is another type of injury producing the same clinical signs of which I have spoken, I do not know that we can definitely say the fracture is there simply because we have a cochleo-vestibular paralysis unless we can get an autopsy. Ulrich presented a very useful study of the question a number of years ago, and showed that the eighth nerve can be so badly injured before entrance into the internal auditory meatus that it can produce the same clinical picture as fracture can produce. So if we cannot show the fracture in the x-ray, we cannot say whether it is there or not, but I would say offhand that probably 50 per cent of the transverse fractures can be shown by x-ray, although in the longitudinal type the percentage is much smaller.

Abstracts of Current Articles.

NOSE

Nasal Accessory Sinuses as Foci of Infection in Arthritis.

Williams, Henry L., and Slocumb, Charles H. (Rochester, Minn.), Arch. Otolaryng., 29:829 (May), 1939.

One finds considerable difference of opinion in the literature in regard to the importance of infection of the nasal sinuses as a foci of infection in infectious arthritis. In order to come to a clearer understanding of this problem, the authors examined an unselected group of 100 cases of infectious arthritis. Out of this group, 20 cases of clinically proved sinus disease were found. Of the cases which had developed sinus disease, 12 patients were operated upon. From this study the following conclusions were drawn:

1. Sinusitis is not the principle focus of infection in cases of infectious arthritis.

2. If disease of the sinuses is found, attempts should be made to eradicate it.

TOBEY.

Ethmoid Labyrinth: Anatomic Study, With Consideration of the Clinical Significance of Its Structural Characteristics.

Van Alyea, O. E. (Chicago), Arch. Otolaryng., 29:881 (June), 1939.

This article deals with a detailed anatomical study of 100 specimens. The various cell groups, the growth and the extension, the drainage of the cells, and the surgical and clinical application of the knowledge obtained from this study is discussed.

TOBEY.

Osseous Affections of the Maxillary Sinus.

Collins, E. Godfrey (Aberdeen), J. Laryng. & Otol., 54:121 (Mar.), 1939.

Lesions of the bony wall of the antrum are classified and discussed with several case reports accompanied by x-rays and photographs of gross and microscopic material. All types of periostitis resulting in hyperostosis are grouped under the heading of Leontiasis

Ossea, a departure from the usual classification which might tend to confuse rather than simplify matters both from the clinical and pathological viewpoint.

Tumors and cysts of dental origin are differentiated from those arising from the superior maxilla itself, as are tumors arising from bone and tumors undergoing secondary ossification.

DEAN, JR.

Charlin's Syndrome (Nasociliary Neuralgia). (Sur le syndrome de Charlin.)

Magabeira-Albernaz, P. (Campinas), Rev. de L. O. R., 60:228 (Mar.), 1939.

This is a good account of the combination of inflammation of the anterior segment of the eye, usually with corneal ulceration; severe oculo-orbital pain, and rhinorrhea, described by Charlin in 1930. Due to involvement of the nasociliary nerve, it must be differentiated from sphenopalatine neuralgia and from the pain of acute sinusitis. Any one of the three factors may predominate. Relief is obtained by high local anesthesia in the nose; septal deformities or pressure on the nerve in the anterior ethmoid canal are blamed for most of the cases.

FENTON.

PHARYNX

Bismuth Therapy in Acute Tonsillitis and Pharyngitis (Estado atual da bismutoterapia nas anginas agudas).

Monteiro, A. (Rio de Janeiro), Rev. Brazil O. R. L., 7:121 (Mar.), 1939.

The author reports on his use of soluble bismuth in more than a thousand cases of severe streptococcic tonsillitis and pharyngitis since 1933. Deltoid or gluteal injection of one 2 cc. ampule, followed next day by 1 cc., is usually sufficient to stop pain and fever, reduce swelling and cause exudates to disappear in 24 to 48 hours.

Bismuth does not arrest an established peritonsillar abscess and is of no value in staphylococcic or Vincent's infections.

Monteiro presents favorable reports from numerous South American sources, also from various German and Austrian clinicians, including Ruttin.

FENTON.

LARYNX

Technic of Laryngofissure for Cancer of the Larynx.

Myerson, Mervin C. (New York), Arch. Otolaryng., 29:662 (Apr.), 1939.

The article consists of a brief historical discussion of the operative procedure of the larynx followed by the author's indication of technic and after-care of laryngofissure for cancer of the larynx. The criteria used for selection of cases for laryngofissure are as follows:

1. Cancer limited to one side.
2. Movement of that side unimpaired.
3. No involvement of the arytenoid area.
4. No involvement above the ventricular band.
5. No involvement below the crest of the cricoid cartilage.

Some of the author's observations are as follows:

Anesthetization of the larynx before operation is of definite value. A smaller incision than is usually employed will suffice.

Tracheotomy is not necessary.

Hemostasis is obtained by the use of small sections of dental roll or on the end of fine hemostats. Suture ligatures or plain ties are not used.

Gentle and careful handling of the endolaryngeal tissues is essential.

The laryngologist who is confronted with the necessity for performing a laryngofissure need not be too greatly concerned about the procedure, because it is a relatively simple operation.

TOBEY.

Radiography of Laryngeal Cancer (Posibilidades radiograficas en el cancer de la laringe).

Regules, P., and Canbarrere, N. L. (Montevideo), An. O. R. L., Uruguay, 8:2:85.

This excellent monograph is abundantly illustrated with radiographs, each with an explanatory diagram. Technique recommended is the lateral projection for superior and for anterior subglottic growths; but antero-posterior planigraphy is necessary for lateral subglottic lesions and those involving the median and lower part of

the larynx. With epiglottic cancer, one may study the depth of invasion down and forward into the tongue, indicating removal of the hyoid bone and base of the tongue with the epiglottis, possibly saving the larynx proper. Radiography is unnecessary in cancer of the aryepiglottic fold. It permits definition of the lower limit of the growth when the pyriform sinus or the transition fold from pharynx into larynx are involved. Anterior subglottic cancer, seen in the crico-thyroid triangle and below the edge of the thyroid cartilage, can be identified as to its downward extension.

Planigraphic examination (tomography) permits comparison with the sound side and defines downward limits for anterior or lateral growths. With cancer of the cords, one can determine whether the ventricle has been invaded and whether, depending on extent, cordectomy or radiotherapy is indicated. Similar help is given in lesions of the false cords.

FENTON.

The History of Cancer of the Larynx.

Thomson, Sir St. Clair (London), J. Laryng. & Otol., 54:61 (Feb.), 1939.

The following are significant excerpts from the author's comprehensive paper:

Cancer of the larynx has increased both actually and relatively in the last twenty years.

Treatment by radium and x-ray therapy has been widely studied. Unfortunately, the type of laryngeal cancer in which radium promises most (i. e., intrinsic cancer limited to one cord) is just that in which the lasting benefit of surgery is well established.

(In speaking of x-ray therapy.) In all cases where surgery can hold out good promise of lasting cure, it must still be given the preference.

DEAN, JR.

Pneumothorax and Mediastinal Emphysema Complicating Tracheotomy.

Michels, Merrill W. (Detroit), Arch. Otolaryng., 29:842 (May), 1939.

Pneumothorax and mediastinal emphysema following tracheotomy are much more common, the author feels, than literature would suggest. The mechanisms by which entry of air takes place in the pleural cavity are discussed. Six cases of acute laryngeal obstruction are presented. The mortality of these cases was 50 per

cent. Pneumothorax and mediastinal emphysema occurred in five cases following tracheotomy. In two of the above five cases there was complete recovery with no primary treatment of the pneumothorax. In one of the cases closed drainage was used. One patient died after a single inadequate pleural aspiration, one after repeated pleural aspirations and still another without pleural aspiration.

The author concludes that it is essential to recognize and institute early adequate treatment of these conditions if the patient is to recover. He advises continuous closed drainage rather than repeated pleural aspiration, prompt use of x-rays for diagnosis and the use of the bronchoscope prior to tracheotomy will eliminate many of the dangerous complications mentioned.

TOBEY.

EAR

Physiology of the Vestibular Labyrinth.

Hyndman, Olan R. (Iowa City), Arch. Otolaryng., 29:759 (May), 1939.

This lengthy monograph is a companion paper to the case report "Torticollis Spastica: Suggested Etiologic Relation to the Vestibular Apparatus: Report of a Case." From the author's experience he suggests that there is a relation of torticollis to the vestibular apparatus. In the above monograph there is an extensive review of the vestibular physiology in order that one may understand the pathological function to which the apparatus when defective gives rise and the possible role in the development of torticollis spastica.

The presentation is given according to the following outline:

I. Physiology.
   A. Experimental observations on animals.
   B. Observations on man.

II. General Comment.

III. Special Problems.
   A. Physical nature of the inner ear.
   B. Role of the cerebellum.

The author feels that there is much yet to be done on the function and physiology of the vestibular labyrinth before we shall be able to explain many of the signs which arise from the so-called involvement of this apparatus.

TOBEY.

Torticollis Spastica; Suggested Etiologic Relation to the Vestibular Apparatus: Report of a Case.

Hyndman, Olan R. (Iowa City), Arch. Otolaryng., 29:927 (June), 1939.

The writer has studied very carefully the relation of the vestibular apparatus to spasmodic torticollis in a previous article entitled "Physiology of the Vestibular Labyrinth" from the Archives of Otolaryngology, May, 1939. The present writing is more or less a follow-up of the above mentioned article and contains a report of a case of spasmodic torticollis.

The conclusions drawn from this case are:

Certain cases of spasmodic torticollis have a patho-physiologic basis in the vestibular apparatus.

Evidence appears to indicate that the locus operandi is central.

Elimination of a pathologic end-organ in this case failed to abolish the torticollis permanently.

TOBEY.

Surgical Management of Otogenous Abscess of the Brain.

Adelstein, Leo J. (Los Angeles), Arch. Otolaryng., 29:624 (Apr.), 1939.

The author presents a short review of the literature dealing with the treatment of otogenous abscess of the brain, followed by a discussion of pathogenesis, pathology, diagnosis and treatment.

Three cases of otogenous abscess of the brain with recovery following conservative treatment are reported. Two of the patients had abscess of the temporal lobe. One patient had an abscess of the cerebellum. In all three cases the abscess was secondary to otitis media and mastoiditis. An abscess of the brain is not an acute surgical emergency and, therefore, due time should be allowed for careful neurological study in order that the abscess may be localized and to be sure that the process of encapsulation has had time to take place. The optimum time for surgical intervention appears to be from four to six weeks after initial cerebral involvement.

The author agrees with the others that the highest percentage of recoveries has followed drainage by simple methods by which excessive trauma and spread of infection are avoided. Suggested technic for drainage of brain abscesses is discussed in some detail.

TOBEY.

A New View of Deafness in Otosclerosis and a Possible New Method of Treatment (Eine neue Auschauung über die Schwerhörigkeit bei Otosklerose und vielleicht ein neuer Weg zu ihrer Behandlung).

Meyer, Professor Max (Ankara), Acta Oto-Lar., 27:1 (Jan.-Feb.), 1939.

In six cases of progressive, severe deafness, of which four were typical otosclerotics, immediate and startling improvement with loss of head noises took place when cerebrospinal fluid was removed by suboccipital puncture. Hearing returned while the fluid was coming out under pressure and has remained six months; apparently decompression of the labyrinth occurred. Meyer suggests the possibility of revision of the etiology of deafness ordinarily termed otosclerotic, and asks others to confirm the findings of this preliminary and startling report.

FENTON.

Congestion of the Artery of the Handle of the Malleus in Hypertension (Das Zeichen der Hammergriffsarterie im Bilde der arteriellen Hypertension).

Monteiro, A. (Rio de Janeiro), Acta Oto-Lar., 26:1:57 (Jan.-Feb.), 1938.

Intense congestion of the artery running along the handle of the malleus has been observed by Monteiro since 1930, in many cases complaining of tinnitus, dizziness and a feeling of distress in the head, with normal hearing and negative nose and throat findings. Blood pressure was found very high in all such individuals. He suggests that this sign (congestion of the artery) should be watched for in all cases of otherwise unexplained vertigo and tinnitus.

FENTON.

Otogenic Meningitis.

Cawthorne, Terence (London), J. Laryng. & Otol., 54:444 (Aug.), 1939.

The author presents an exhaustive study of otogenic meningitis, including his views of therapy in the light of his experiences with sulfanilamide and similar drugs. He feels that chemotherapy plays the predominant part in the management of otogenic meningitis. When there is evidence of diseased bone in contact with the meninges, it should be removed. When there is no evidence of diseased bone in contact with the meninges, however, surgical procedures other than paracentesis should be omitted and the therapy restricted to chemotherapy, drainage of spinal fluid, vaccines and transfusions.

The author condemns the injudicious use of intrathecal serum and drugs, but believes that 0.8 per cent sulfanilamide is probably safe.

A detailed table of sixteen cases with their findings, treatment and results is presented.

DEAN, JR.

Otitis Media and Mastoiditis Simulating Thrombophlebitis of the Lateral Sinus.

Ersner, Matthew S. (Philadelphia), Arch. Otolaryng., 29:605 (Apr.), 1939.

When sepsis occurs with suppuration of the temporal bone, one is led to believe that phlebitis and thrombosis of the lateral sinuses are responsible for sepsis in 100 per cent of the cases. However, actually the incidence of sinus thrombosis and phlebitis in otologic diseases is only 2 per cent. To illustrate how easily one may be misled by symptoms and signs of sepsis, the author cites five cases. Any case of sepsis is entitled to a deliberate and deep consideration from a medical and surgical standpoint. Because of the ease with which mistakes are made, the surgical treatment of the lateral sinuses should not be an emergency measure but should be instituted only after a long period of conservative treatment and long deliberation.

TOBEY.

Books Received

Otolaryngology In General Practice.

By Lyman G. Richards, M.D., Fellow in Otology, Courses for Graduates, and Assistant in Surgery, Harvard Medical School; Associate Professor of Otolaryngology, Tufts Medical School. Cloth. 8vo. of 352 pages with 72 illustrations. The Macmillan Company, New York, 1939.

With his "Otolaryngology in General Practice," Richards supplies the American practitioner with the type of volume until now more familiar in Europe than on this continent. The English doctor, for example, treats nose and throat cases as a matter of course. Only when the diagnosis is in doubt or the surgery forbidding is the specialist consulted. After the consultation, the patient commonly reverts back to the "G. P." for treatment.

As a consequence of this system, there exists in England a plethora of small ear, nose and throat manuals for general practitioners. Perhaps with less candor but no less persistence, the American practitioner through choice or force of circumstances retains what he can of special work, which is scarcely surprising in the light of Richards' estimate that some 20 per cent of the general practitioner's work is with diseases of the upper respiratory tract.

Recognizing the need of an authoritative work to guide him, and to help him to distinguish between work which he is qualified to undertake and that which may be disastrous in his hands, Richards has followed in the footsteps of his distinguished father, who produced a similar work a quarter of a century ago. He has succeeded admirably in his stated purpose, maintaining completeness without confusing detail. He has adhered, in his descriptions, largely to methods which he finds worthy, and while some teachers will disagree, the audience to which this book is directed will no doubt be better served by a single point of view than by having to choose between several.

The book is engagingly written, clear and concise, and complete. The illustrations are a delight.

The Mechanism of Thought, Imagery, and Hallucination.

By Joshua Rosett, M.D., Professor of Neurology in Columbia University; Scientific Director, Brain Research Foundation, Inc., New York. Cloth. Royal 8vo. of 289 pages. Columbia University Press, New York, 1939. Price, $3.00.

Occasionally a volume appears which, although it has no direct bearing upon the subject of laryngology, is nevertheless of interest to the specialist as a physician and a scientist, and on that basis finds a place here. Such a book is "The Mechanism of Thought" by Rosett.

To those who are not psychologists and physiologists, this work has a special appeal, because it lays down its thesis in plain language and develops it in a fashion which any educated person can understand.

Beginning with a discussion from the modern viewpoint of the factors surrounding Hughlings Jackson's Law of Evolution and Dissolution of the Nervous System, and the James-Lange theory of the relationship of emotions to bodily states, the author draws upon his observations of the will, of the mental short-hand of Symbolism and of injuries to the complex machinery of the memory.

He then presents his views on the mechanisms of hallucinations and of epileptic seizure which, he states, "is on the whole the same as that which is operative in the several stages of each state of attention, and in those of sleep."

He concludes with an absorbing chapter on attention and one on sleep.

He points out, among other conclusions, that the function of thought is concerned with the subjective reproduction and orientation in the past and at the same time in the present, and that increasingly smaller amounts of orientation in the present permitting more vivid and at the same time less accurate relation of elements from the past, constitute imagery and hallucination.

The Art of Anaesthesia.

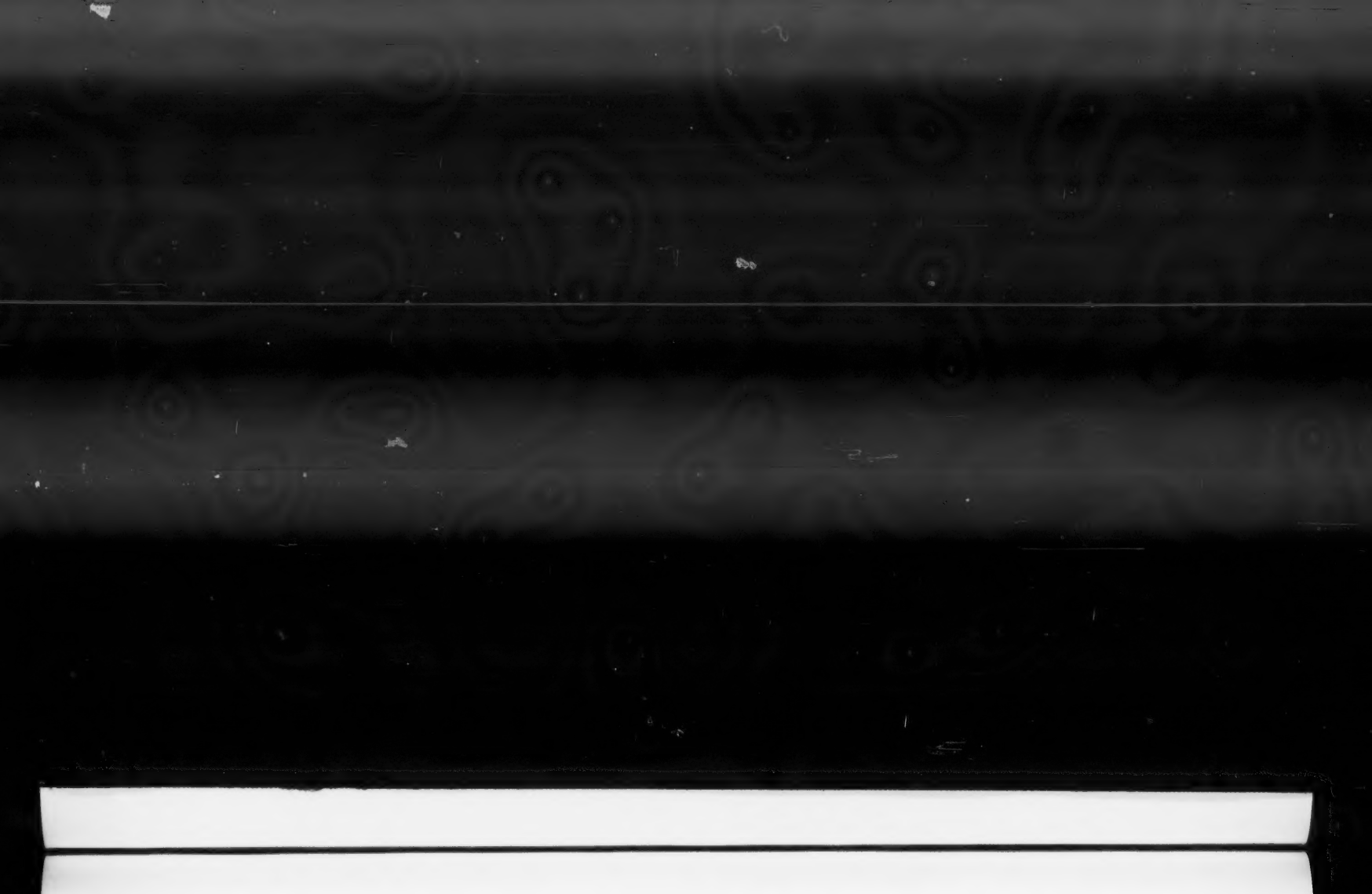
By Paluel J. Flagg, M.D., Visiting Anaesthetist to Manhattan Eye and Ear Hospital; Consulting Anaesthetist to St. Vincent's Hospital, New York, Woman's Hospital, Sea View Hospital, Jamaica Hospital, Mount Vernon Hospital, Flushing Hospital, Mary Immaculate Hospital, St. Mary's Hospital, Far Rockaway, N. Y., Nassau Hospital, L. I.; Director of Pneumatology, World's Fair, New York City, and Chairman of Committee on Asphyxia of the American Medical Association. 8vo. of 491 pages with 161 illustrations. Sixth Edition Revised. J. B. Lippincott Company, Philadelphia, 1939.

Another book from a neighboring speciality, "The Art of Anaesthesia" by Paluel J. Flagg, is worthy of study.

Few persons are less concerned with the vagaries of anaesthetics, apparatus and anaesthetists than is the laryngologist, chiefly for the reason that he is usually occupying the center of the field which the anaesthetist normally requires for his ministrations. Accordingly, the latter often works under conditions somewhat outside his usual routine, and in the presence of blood and secretions, gags and other instruments.

In view of the important bearing which the skill of the anaesthetist may have upon the patient's postoperative comfort and safety, and hence upon the reputation of the operator, the surgeon may with much profit familiarize himself with the problems of his colleague by the perusal of such a book as this.

Eye, Ear, Nose and Throat Manual For Nurses.

By Roy H. Parkinson, M.D., F.A.C.S., Head Oculist and Aurist to St. Joseph's Hospital, San Francisco, Cal. Crown 8vo. of 243 pages with 79 illustrations. Fourth Edition. The C. V. Mosby Company, St. Louis, 1939.

This verdant little volume (the paper is green!) is somewhat less regional in its appeal than most books of this type. It is brief to a degree, designedly so, as the author states in his preface. The half-tone illustrations on the whole are good. The line drawings and the audiogram are incredibly messy and confusing. A physician encountering such an illustration can lay it aside and consult his anatomical atlas; the poor nurse whose otolaryngological reference library may consist of one book deserves to find in it accurate and legible illustrations and diagrams.